Bulletin No. 11 of the Department of Surgery
University of Lund, 221 85 LUND
1977

SUBCUTANEOUS TRANSPOSITION OF THE SPLEEN

An experimental and clinical study

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Akademisk avhandling
som för avläggande av medicine doktorsexamen
vid medicinska fakulteten vid Universitetet i Lund
kommer att offentligen försvaras å Föreläsningssal 3,
Centralblocket, Lasarettet i Lund onsdagen den 18 maj
1977 kl. 09.15

From the Departments of Surgery and Diagnostic Radiology,
University of Lund, Lund, Sweden.

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by

Bengt Börjesson

Lund 1977

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BIBL.

The present thesis is based on the following papers:

I. S. Bengmark, B. Börjesson and T. Olin. Development of porta-systemic shunts after subcutaneous transposition of the spleen in the rat. Am. J. Surg. 125:757, 1973.

II. S. Bengmark, B. Börjesson and T. Olin. Development of porta-systemic shunts after subcutaneous and extraperitoneal transposition of resected spleen. A comparative study in the rat. Acta Chir. Scand. 141:739, 1975.

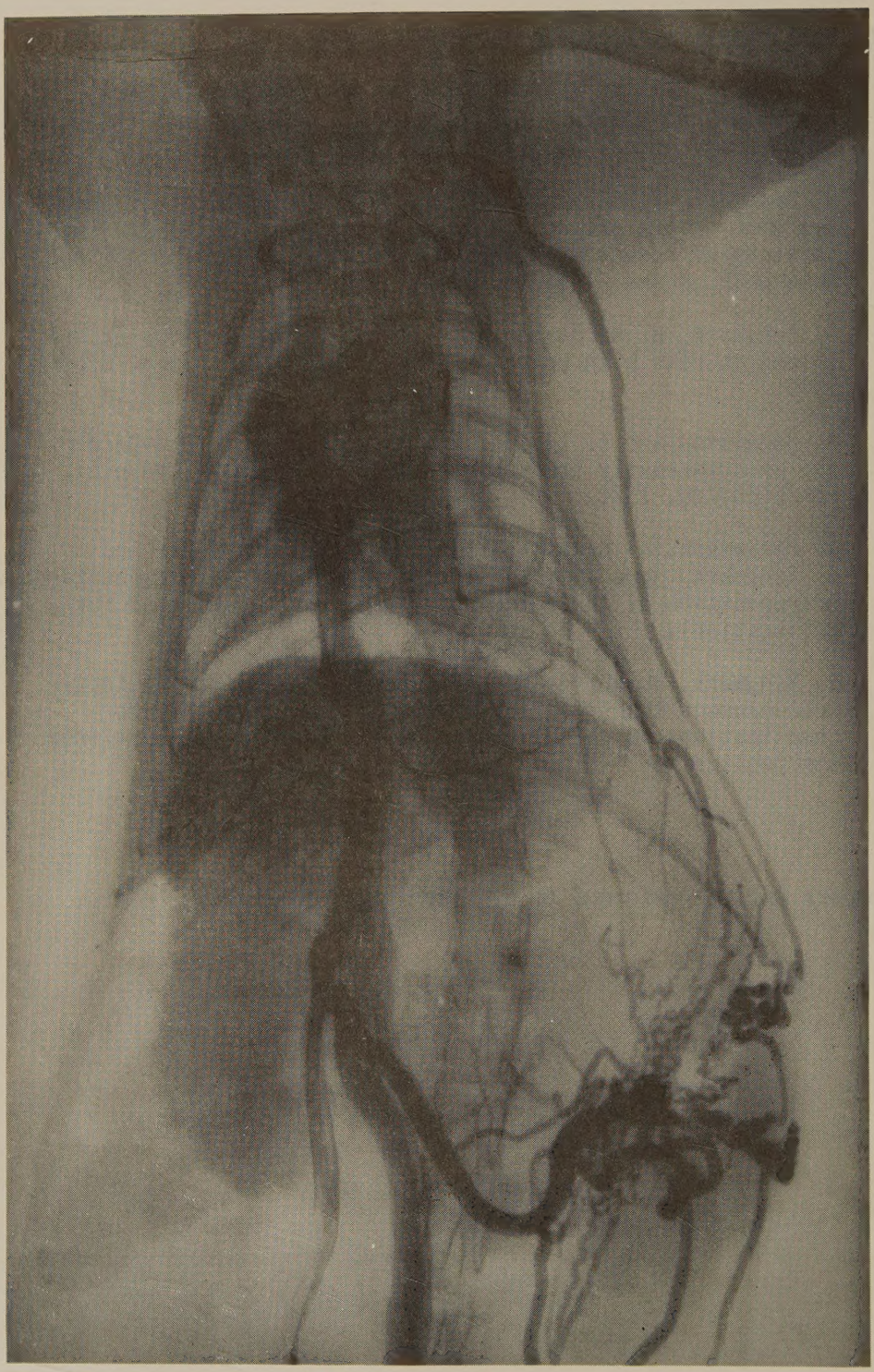
III. S. Bengmark, B. Börjesson and T. Olin. Transposition of the spleen in rats with portal hypertension. Br. J. Surg. 63:268, 1976.

IV. B. Börjesson and I. Idvall. The microscopical appearance of the subcutaneously transposed spleen. Ann. Chir. Gynaecol. Fenn. 65:220, 1976.

V. B. Börjesson, E. Cavallin-Ståhl, B. Lund, C. Mercke and S. Bengmark. Heme catabolism studies in rats after subcutaneous transposition of the spleen. J. Surg. Res. (Submitted for publication).

VI. S. Bengmark, B. Börjesson, A. Lunderquist and B. Sigstedt. Subcutaneous transposition of the spleen - A method for complications in portal hypertension? Ann. Surg. (Submitted for publication).

The papers will be referred to by their Roman numerals, I - VI.



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"If I, for example, were bleeding from esophageal varices, I should like to be treated at an academic institution, preferably one where a controlled investigation was in progress. I would try to meet the criteria for inclusion, since the prognosis of those who do is much better than for those who do not. I would fervently hope to be selected - randomly, of course - for the operative group. Then, in the light of previous findings, and with faint heart, I would refuse surgery."

Harold O. Conn 1974.

INTRODUCTION

Obstruction of the portal venous flow results in increased pressure in the portal vein system - portal hypertension. In adults the cause of this obstruction is most often a liver disease and more seldom an occluding process in the extrahepatic portal vein. In children the conditions are reversed. Among liver diseases causing portal hypertension cirrhosis of the liver is in Sweden totally dominating. In 1974 liver cirrhosis caused 859 deaths in Sweden. This is about the same figure as for death from rectal cancer and 150 more than the death from leucemia (Central Bureau of Statistics, Stockholm, 1976). In most instances there are very limited possibilities to influence the cirrhotic process and unfortunately the therapeutical efforts more often have to be focused on the complications of portal hypertension than on the primary disease. Among complications often seen in patients with portal hypertension esophageal varices, ascites, hepatic encephalopathy and hypersplenism are of the greatest clinical significance. In most cases ascites is today well-controlled by modern diuretics, encephalopathy can be controlled or at least relieved by dietary and pharmacological treatment and hypersplenism is seldom of such severity that it as such significantly affects the life of the patient. The remaining and greatest problem is the esophageal varices and bleeding from these is a constant threat to the patient's life.

Incidence and clinical significance of esophageal varices.

The incidence of esophageal varices among patients with liver cirrhosis has been attributed a great variation of figures. According to Voorhees (1974) about 12 percent of patients with liver cirrhosis will develop demonstrable esophageal varices. Among 387 consecutive cases with alcoholic cirrhosis Snyder et al (1975) reported varices in 44 percent. They found that the patients with varices were older and had had cirrhosis of longer duration than those without varices. Brick and Palmer (1964) found endoscopically esophageal varices in 70 percent during initial esophagoscopic examination of 1000 patients with liver cirrhosis. In an autopsy material from Gothenburg consisting of 244 cases with liver cirrhosis Olsson (1972) reported esophageal varices in 61 percent.

However, many patients with esophageal varices will never bleed from them. In four different prospective randomized studies of the prophylactic portacaval shunt the rate of hemorrhage in the medical control-group was 19 percent during the average of 42 months in the Veterans Administration Cooperative Study (Jackson et al 1968); it was 27 percent in the Boston Interhospital Liver Group Study which extended over ten

years (Resnick et al 1969); it was 39 percent in the study from West Haven extending over 57 months (Conn and Lindenmuth 1968) and 27 percent in another study reported by the same group and extending over six years (Conn and Lindenmuth 1972). Similar figures have been reported in other studies - 29 percent during an average period of follow-up of 67 months (Snyder et al 1975) and 29 percent during an average follow-up period of 39 months (Baker et al 1959). Although most of those patients who bleed from esophageal varices do so within two years after the varices are discovered and then with decreasing frequency (Baker et al 1959, Conn et al 1972), it is reasonable that frequency figures in a certain material will increase with the time of follow-up. In the above-mentioned autopsy material from Gothenburg (Olsson 1972) 67 percent were considered to have bled from the varices intra vitam.

Estimates of mortality from exsanguination during the initial variceal hemorrhage vary from ten percent (Baker et al 1959) to 75 percent (Cohn and Blaisdell 1958). Summing up different series it has been concluded that about 50 percent of the patients will succomb to progressive liver failure or exsanguination or a combination of both following each instance of bleeding, including the first (Voorhess 1974).

As a final cause of death among cirrhotic patients hemorrhage has been considered to be the main reason from 11 percent (Hällén and Krook 1963 - Swedish series) to 34 percent (Garceau and Chalmers 1963 - U.S. series).

PURPOSE AND PLAN OF THE PRESENT STUDY

When this study was initiated portacaval shunting had been extensively used in the treatment of portal hypertension for about 25 years. Its advantages as well as its disadvantages were well-known. It was evident that a successfully performed shunting almost abolished the risk for new variceal bleeding and it was also obvious that ascites was in most cases effectively treated by the shunt. However, already in the fifties there was criticism about the shunting surgery, especially expressed by Nachlas (1958). He proposed that a marked reduction in portal flow would sustain further injury to the liver and he also doubted if the prognosis of the patient undergoing portacaval decompression would be improved. In 1970 a 26-year experience was published, reviewing the results of 404 cases of portal hypertension in adults with liver cirrhosis who had been subjected to portacaval shunting (Voorhees et al 1970). Encephalopathy was found to be the major problem and the chief reason why many patients with otherwise successful shunts were unable

to re-enter a productive life. Incidence of moderate and severe encephalopathy combined was 34 percent in the follow-up period. Similar figures for encepahlopathy were also found in England where Hourigan et al (1971) reported severe chronic encephalopathy in 38 percent of patients surviving elective shunt surgery. At about the same time it was also clear from the four prospective controlled clinical trials of prophylactic shunting mentioned above that although the shunting very clearly reduced the risk of rebleeding, it did not prolong life. It was evident that the shunts caused an increased rate of death in liver failure and an increased frequency and a more severe degree of liver encephalopathy (Jackson et al 1968, Conn and Lindenmuth 1968, Resnick et al 1969 and Conn et al 1972).

It was obvious that the surgical treatment of portal hypertension lay far from its final solution. Various methods have been proposed to bring about selective decompression of the esophagogastric region and at the same time maintaining high pressure in the portal vein: Warren et al 1967 suggested a distal spleno-renal shunt, LeVeen et al 1970 a cavo-splenic shunt and Inokuchi et al 1970 a left gastrivenous-caval shunt. Britton et al (1970) suggested a side-to-side spleno-renal shunt. After central ligation of the splenic vein even this shunting procedure can be regarded as resulting in selective decompression. At least in the two first-mentioned series the mortality rates were discouraging. Hästbacka published in 1971 the results of thoracic transposition of the spleen in 37 patients with portal hypertension. The idea was that the patient should develop portasystemic collaterals in a region where the risk of bleeding was small. Because of the vicinity of these collaterals to the esophageal varices it was suggested that the method could have selective decompressive properties. Although it was clear that this procedure was not as effective as the portacaval shunt in preventing recurrent bleeding it was also evident that it did not cause encephalopathy or aggravation of the liver damage. However, some patients had intra-thoracic complications consisting of pleuro-cutaneous fistula, hemothorax and restrictive ventilatory impairment. Regarding these facts we decided to investigate the possibilities to achieve the same collateral development by a subcutaneous transposition of the spleen hoping to avoid the thoracic complications mentioned above. If complications occurred they would probably be more easy to handle subcutaneously than in the thorax.

Before attempting subcutaneous transposition of the spleen as an alternative clinical method for the treatment of portal hypertension we needed the answers to the following questions given below. For this purpose animal experiments were used.

1. Do portasystemic shunts develop after the proposed operation and can this development be influenced by modifications in the surgical technique?

2. Do the developed collaterals have the capacity to shunt a substantial portion of the portal flow?

3. Does the operation significantly influence life of the animal?

These three questions were studied in the first paper (I).

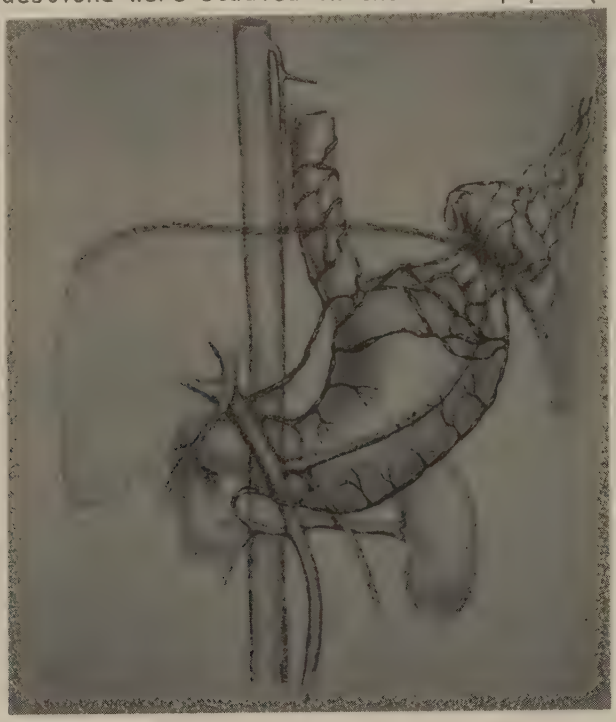

Fig. 1. Illustration of portasystemic collateral development after thoracic transposition of the spleen.

From the first study arose the following questions:

4. Can the same collateral development be achieved after transposition of a resected spleen? Resection seemed to be a prerequisite for clinical application.

5. How can the spleen be resected without comprimising the collateral development too much?

6. Can the shunting be just as effectively produced by simple extraperitonealization of the spleen?

These questions were studied in the second paper (II).

In the third paper (III) we evaluated the operation in portal hypertensive animals concentrating mainly on the following two questions:

7. Do the collaterals relieve induced portal hypertension?

8. Are there a priority of collateral flow in the parasplenic collaterals in relation to collaterals spontaneously developed elsewhere?

The influence on the histological picture of the subcutaneous localization of the spleen was the objective of our fourth paper (IV):

9. Will the splenic tissue atrophy when located subcutaneously?

The purpose of the fifth paper (V) was to study hematological consequences of the operation:

10. Will a subcutaneous location of the spleen and an increased shunting of blood over it cause hypersplenism? If so, it can possibly be of clinical significance in patients with liver cirrhosis who already have a decreased red cell life span (Jandl 1955, Hall 1960).

In the sixth paper (VI) our clinical observations are reported.

MATERIAL AND METHODS

The experimental studies (I-V).

Male Sprague-Dawley rats were used. During the experimental periods they had free access to water and standard food pellets. Ether anaesthesia was used for the operations and during angiography and pressure measurements. Operation equipment was clean but not sterile. Operative techniques used are described in the respective papers. Collateral development and capacity were evaluated by registration of survival and pressure in the superior mesenteric vein after an acute portal vein ligation and by angiography.

Portal vein ligation (I-V): Through an upper midline incision the portal vein was totally ligated immediately above the pyloric vein using a 4-0 silk suture.

Pressure measurement and portal angiography (I, II and III): A mesenteric vein was punctured with a scalp vein cannula (Miniven small vein set, 21 gauge, Portex, England). The pressure was measured through the cannula with an electromanometer (Elema-Schönander, Sweden). Contrast medium, Isopaque cerebral (meglumine metrizoate, Nyco A/S, Norway), was injected by hand with a syringe and serial pictures were taken at the rate of one picture per second.

Induction of portal hypertension (III).

Portal hypertension was in one way induced by progressive occlusion of the portal vein with ameroid constrictors (Threepoint Products Co., Canada). These are rings of a hygroscopic casein derivative encased in stainless steel; as water enters the casein the lumen diminishes (Berman and Judy 1955). With the size of rings used in this experiment the portal vein was occluded after about one week and the portal pressure was increased to twice the normal value (Liehr et al 1971). In another way portal hypertension was elicited by intragastric administration of 2.5 ml of a solution of dimetylnitrosamine (DMNA; 500 p.p.m.) once a day for three consecutive days per week over six weeks. This substance is known to produce liver damage with features of human cirrhosis (Madden et al 1970, Haney et al 1972).

Histological examination of the subcutaneous spleen (IV).

All spleen specimens were immediately fixed in ten percent buffered formalin. Thin slices of the spleens were then embedded in paraffin blocks from which tissue sections of 4 μ thickness were made. These sections were stained with hematoxylin and eosin and by the modified method of van Giesen for collagen fibres, Gridley's reticulum stain, periodic acid-Schiff reaction, Gomori's iron reaction and Romeis' method for fat staining with scarlet red. The sections were studied in a light microscope.

Evaluation of a possible hypersplenic effect of the operation (V).

Some hematological data from four groups of animals earlier subjected either to subcutaneous transposition of the spleen alone or this operation followed by portal vein ligation were compared to those of an unoperated control group. In this way a possible hypersplenism secondary to an increased collateral flow over the spleen was estimated. At the time of study the rats had been starved for 12 hours and were made unconscious with a neck blow. After laparotomy blood was sampled by aortic puncture. The spleen was perfused with ice cold 0.1 M potassium phosphate buffer (pH 7,4) and removed for the determination of heme-oxygenase activity (Tenhunen et al 1969). Hemoglobin concentration, reticulocyte count and platelet count were determined. The CO content of the blood samples was determined by gas chromatography as described by Collison et al (1968) with the modification by Lundh et al (1972). Direct reacting and total bilirubin concentration was determined according to Michaëlsson et al (1965).

The clinical study (VI).

Eleven patients with portal hypertension were treated with subcutaneous transposition of a resected spleen. In one of the patients portal hypertension was due to a portal vein thrombosis - in the other ten it was due to hepatic disease - cirrhosis in nine and chronic hepatitis in one. Eight of the patients were operated after variceal hemorrhage - from varices in the esophagus in seven and from ileostomal varices in one. These eight patients constituted the "therapeutic group". In three patients the operation was performed without a preceding hemorrhage. They constituted the "prophylactic group". In two patients the main aim of the operation was to reduce the risk of future hemorrhages. The third patient had a severe thrombocytopenia as the main indication for treatment.

A left-hand-sided transrectal incision was used for operation. The peritoneum on the lateral side of the spleen was incised and the spleen and the pancreatic tail were mobilized medially keeping the vascular connections intact with the gastric fundus (the short gastric vessels). A vascular clamp was applied over the hilar vessels. The spleen was resected as shown in fig. 2 and the remaining piece of the spleen with its vascular connections was transposed subcutaneously via a separate subcostal incision (fig. 3).

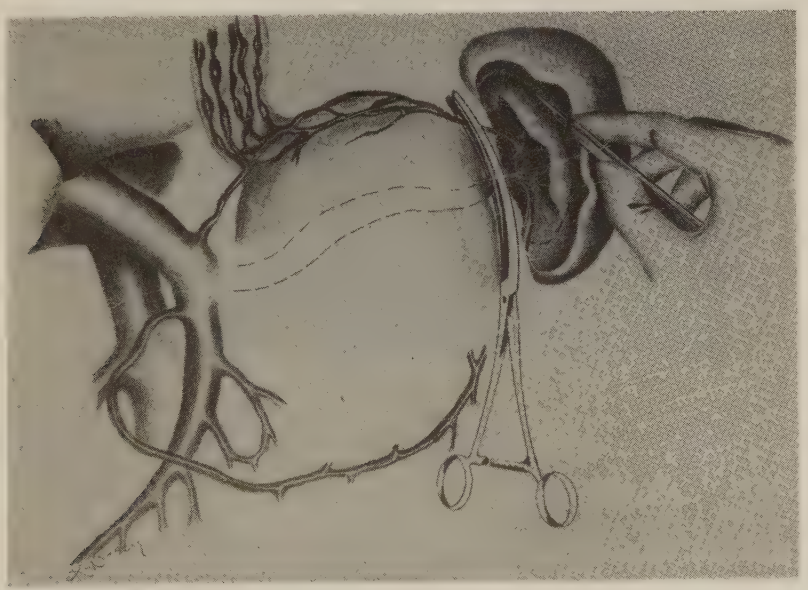

Fig. 2. The spleen has been mobilized and a vascular clamp applied over the hilar vessels. The splenic parenchyma is resected.

Fig. 3. The remaining piece of the spleen is in its subcutaneous position. The greater curvature of the stomach is suture fixated to the adjacent inner abdominal wall. There is a tube for suction drainage in the subcutaneous pouch.

Pre- and postoperatively routine blood and liver tests were carried out. The influence on esophageal varices was evaluated by barium swallow X-ray and by esophagoscopy, the influence on portasystemic collateral flow by study of the venous phase of celiac and superior mesenteric arteriography. Besides a complete history, routine EEG and psychometric testing (Rehnström et al 1977) were used for detection of liver encephalopathy.

RESULTS

The experimental studies I-V.

Influence of modifications in the operative technique on collateral development; shunting potential and effect of the operation on the animals.

The operation resulted in a rapid development of portasystemic collate-

rals via the spleen. They could not be seen angiographically after two days but after five days they were easily demonstrated. After that they did not increase in size significantly. However, after ligation of the portal vein they showed a marked increase in size.

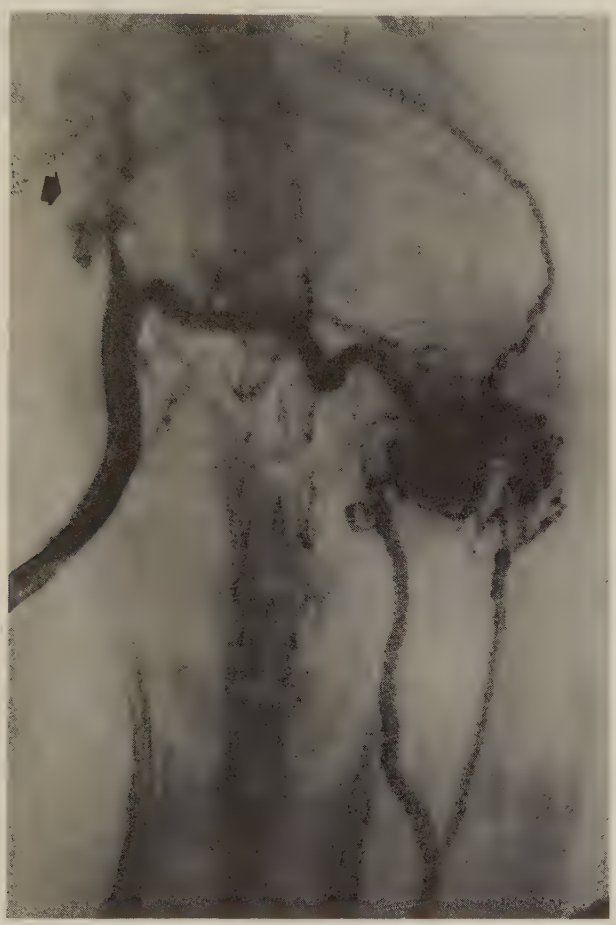

Fig. 4. Portography three weeks after subcutaneous transposition of the spleen and one week after ligation of the portal vein. The portal flow is directed over the splenic vein and over the enlarged parasplenic portasystemic collaterals.

Total portal vein ligation kills the control animal after about an hour because of severe congestion in the bowels. After subcutaneous transposition of the spleen, however, the parasplenic collaterals were already after a week of sufficient size to prevent death after portal vein ligation in about half of the animals. When the portal vein ligature was applied three and six weeks after the transposition

all animals survived when the spleen was transposed via a subcostal incision. The survival rates were not so good after midline incision, lateral subcutaneous dissection and translocation of the spleen to its new position via a small subcostal transmuscular hole made by forceps. No sutures were used around the splenic hilus in the latter modification of the operation. Probably the latter procedure was too atraumatic to promote the development of adhesions and new vascular connections. The survival rates after portal vein ligation indicate a favourable effect on collateral development of irritating substances e.g. talcum applied locally. This effect could also be achieved by splenic decapsulation or bipolar resection.

The pressure measurements in the superior mesenteric vein show a considerable increase in pressure after portal vein ligation. The pressure became progressively normal during the week after ligation, evidently reflecting the concomitant increase in collateral size.

The rats subjected to subcutaneous splenic transposition two years earlier were in very good condition. This indicated that the operation had no major deleterious effects on the experimental animal.

Collateral development after different types of splenic resection and after extraperitonealization of the spleen (II).

As can be seen from our first paper (I) we had the impression that the collaterals originated mainly from the splenic hilus. This was confirmed in paper II. Both water soluble and oil contrast medium were used for angiography. The oil contrast medium was not taken up in the spleen by the reticuloendothelial cells which would have been the case if the splenic parenchyma had to be traversed by the collateral flow. Angiography revealed better collateral development when three segments were transposed instead of one. The difference was most evident after one week. This finding is in accordance with the higher survival rate and lower pressure elevation after portal vein ligation one week after transposition, when three segments were transposed instead of one. When just the hilus region was transposed the degree of collateral development seemed to be somewhere in between the two above-mentioned groups.

Simple extraperitonealization of the spleen was an inferior method judged by angiography and survival after portal vein ligation.

Fig. 5. Portography three weeks after transposition of three splenic segments to the subcutis. V. portae was ligated immediately before the angiography. Good portasystemic collateralization over the splenic hilus.

Fig. 6. Portography one week after extraperitonealization of the spleen. V. portae was ligated immediately before angiography. Very slight collateral development.

Collateral development in portal hypertension and its influence on portal vein pressure (III).

The transposed spleen was shown to be the predominant locale for porta-systemic collateral development in portal hypertensive rats, if the pressure elevation was due to prehepatic as well as to hepatic portal vein obstruction. On the other hand, the control animal with prehepatic obstruction had mainly bridging collaterals over the constricting ring. In the control animal with hepatic obstruction the collaterals were mainly spleno-renal.

Moreover , the collaterals found in relation to the transposed spleen seemed to reduce portal pressure more effectively than those developed spontaneously in the control animals.

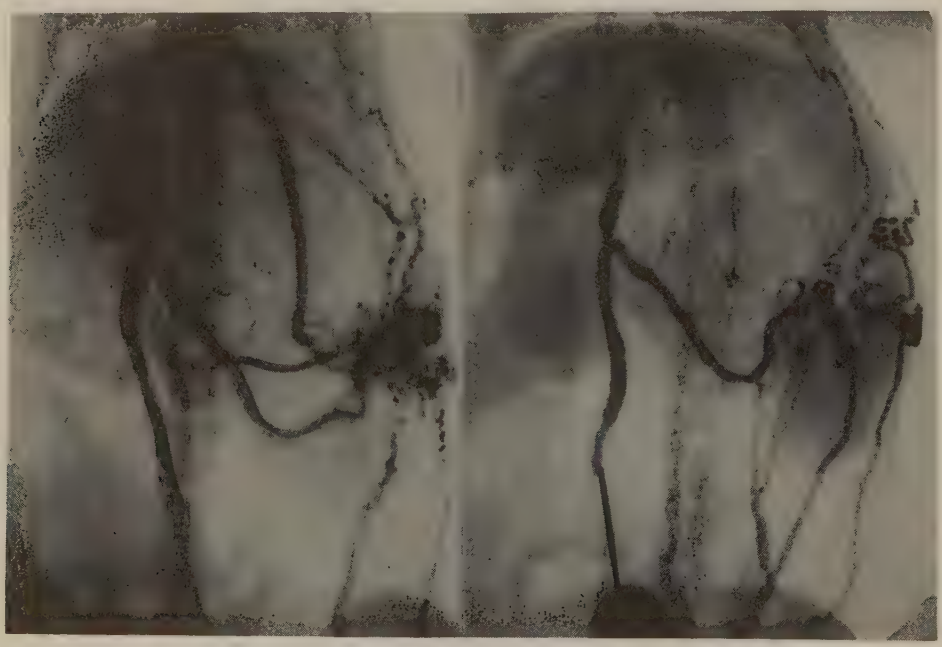

Fig. 7. (to the left) Portogram two weeks after ameroid application and splenic transposition. The collateral flow is predominantly over the spleen. The splenic vein is dilated.

Fig. 8. (to the right) Portogram after DMNA treatment and splenic transposition. The calibre of the portal vein branches are diminished. The dominating collateral flow is via the widened splenic vein.

Influence of the subcutaneous location on splenic histology (IV).

As well as in the experimental as in the clinical part of this study there were signs of congestion with dilated sinuses in the subcutaneous spleens. The splenic capsule was thickened with cellular fibrosis containing dilated capillaries and venules. At some points the sinuses were seen to drain directly into the vessels of the capsule. Apart from the congested red pulp and the capsular fibrosis the subcutaneous position did not seem to give rise to serious histological changes.

Hematological consequences of the subcutaneous location and of the increased shunting over the spleen (V).

The portasystemic shunting over the spleen reported in papers I-III and the congestion found in paper IV could possibly result in hypersplenism. To elucidate this problem methods for evaluating hemoglobin heme turnover were used for study. Two weeks after subcutaneous transposition of the spleen a slight hemolysis was found indicated by increases in COHb percent saturation, reticulocyte count and hemeoxygenase activity in the spleen. Two days after portal vein ligation, resulting in a still more pronounced congestion and collateral flow over the spleen, the hemolysis was more pronounced now also resulting in a decrease in hemoglobin concentration and an increase in serum bilirubin. The increased red cell turnover was gradually normalized after the portal vein ligation. Eight weeks after the ligation there were no signs of hemolysis. Platelet count was slightly reduced two days after portal vein ligation but became normalized again.

Conclusions drawn from the experimental studies.

- A subcutaneous transposition of the spleen in rats results in a rapid development of portasystemic collaterals via the transposed spleen.
- These collaterals are already after two weeks of sufficient size to cause a portasystemic shunting of such magnitude that the animals can stand an acute portal vein ligation.
- The collateral development can be increased by decapsulation of the spleen, by bipolar resection or by application of talcum powder to the splenic surface.
- The collaterals increased markedly in size after portal vein ligation simultaneously with a gradual normalization of portal pressure elevation seen after the ligation.
- The operation had no detrimental effect on the animals.
- The collateral development can also be achieved after transposition of a resected spleen but to a lesser degree after simple extraperitonealization.
- The collateral development is mainly from the splenic hilus. Consequently it is essential to save as much as possible of the hilus when the spleen is resected.

- In animals with prehepatic or hepatic portal obstruction the priority of collateral flow is in relation to the transposed spleen. Furthermore, these collaterals reduce portal pressure more effectively than those developed in the animal not subjected to splenic transposition.
- The subcutaneous splenic tissue shows congestion but no other serious histological changes.
- The subcutaneous location of the spleen and a pronounced portasystemic shunting over it does not cause a persistent hypersplenism.

The clinical study

Effect of the operation on patients with portal hypertension (VI).

Among the three patients operated on without a previous bleeding history - "the prophylactic group" - two died in the postoperative period. One patient died after two days because of uncontrollable intraabdominal bleeding and the other after two weeks in liver failure.

In the "therapeutic group" a 77-year-old woman died after a week in circulatory failure - possibly secondary to an intraabdominal abscess. Two patients in this group had to be reoperated because of intraabdominal bleeding.In connection with one of these operations the remaining piece of the spleen was extirpated in one patient. In two patients the splenic remnant has atrophied completely later on. Consequently only five patients had subcutaneous splenic remnants after a year. In three of them the transposed tissue has remained the size it had after resection and in two it has diminished. The splenic tissue has not been vulnerable in its new position and has not been of cosmetic concern to the patients. Needle biopsies taken from the splenic tissue one and three years postoperatively have shown signs of congestion with dilated sinuses but a lesser degree of fibrosis of the framework as compared to the preoperative picture.

The patients with sizeable splenic remnants have all developed reversed flow direction in the splenic vein and portasystemic collaterals over the transposed splenic tissue. However, these collaterals were not of the magnitude found in the animal experiments and they did not reduce portal pressure.

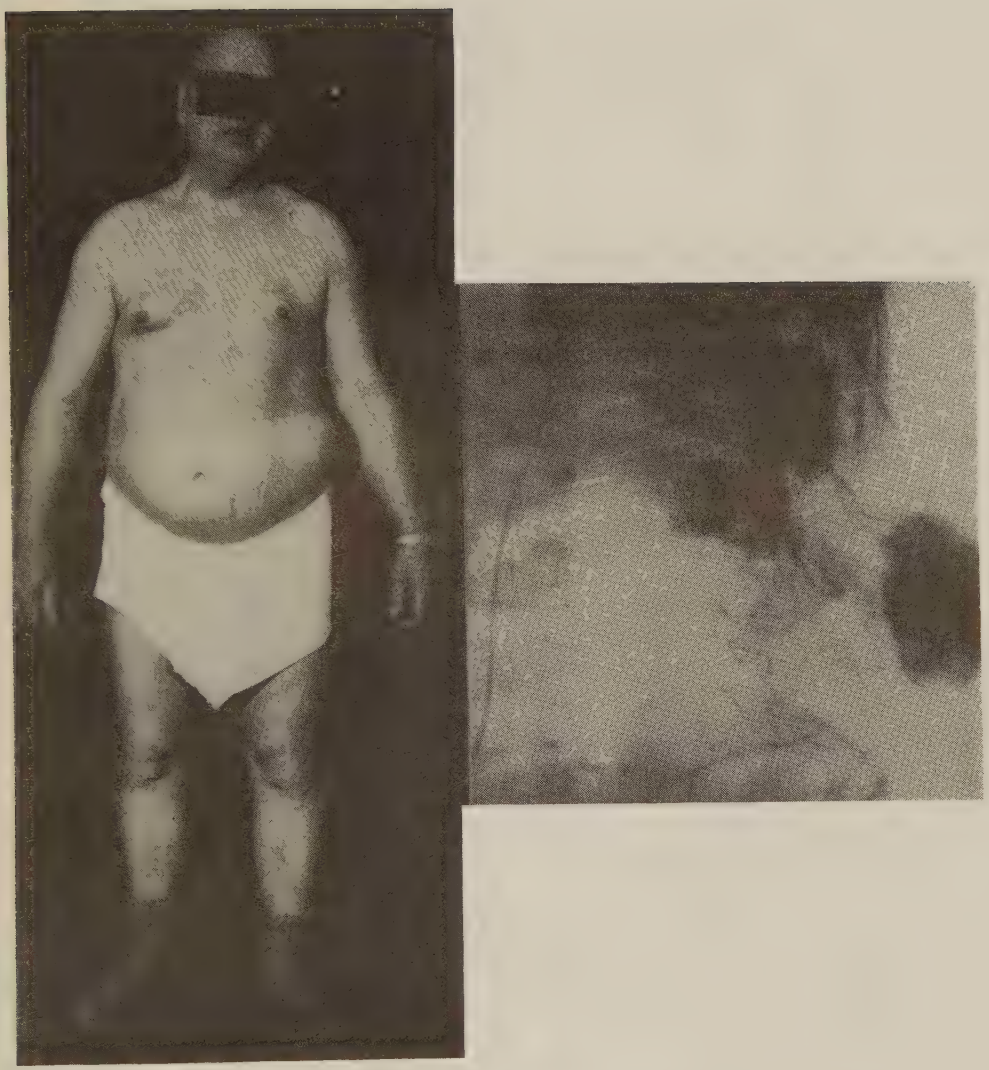

Fig. 9. (to the left) The remaining piece of the spleen is seen as a subcutaneous lump below the left costal margin.

Fig. 10.(to the right) Portasystemic collaterals originating from the subcutaneously positioned splenic remnant in the patient shown in fig. 9. (Venous phase of a celiac arteriography six months after operation).

Despite the remaining portal hypertension only one of the patients with preoperative bleeding from esophageal varices has rebled during a postoperative observation time varying between 44 and 60 months. This patient succumbed to this bleeding. The bleeding locale was never definitely diagnosed. Autopsy showed varices in the gastric fundus and a carcinoma in the cardia. Which of these lesions had caused the bleeding could not be decided.

In only one patient did the esophageal varices disappear postoperatively. In one patient they slightly decreased in size but in the others the operation did not influence the varix size.

The operation has not caused liver encephalopathy and has not been detrimental to liver function. The preoperative signs of hypersplenism have disappeared - even in the patients with sizeable splenic remnants.

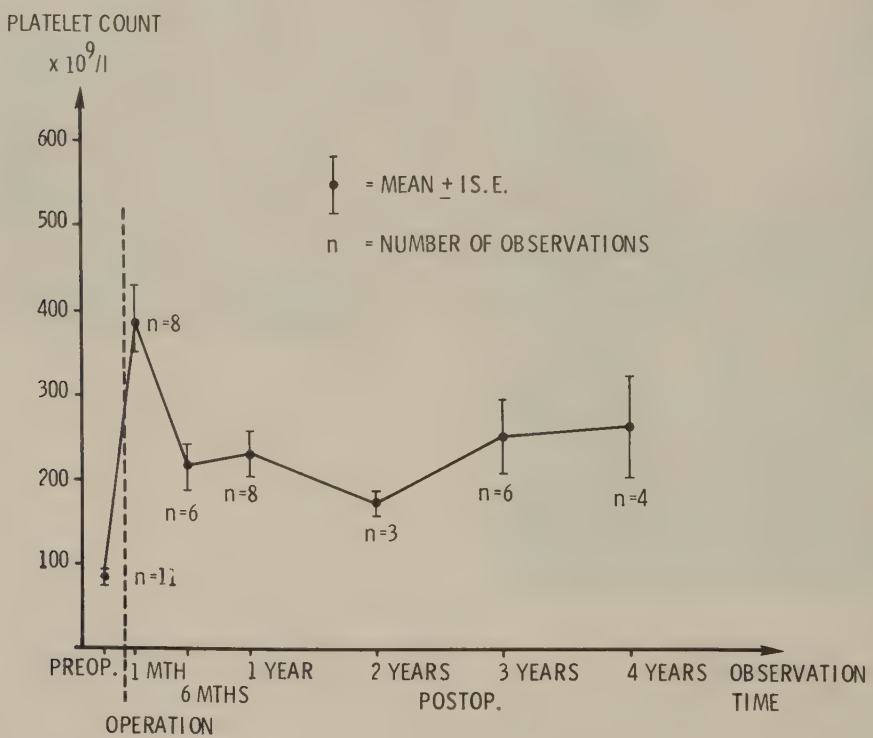

Fig. 11. Pre- and postoperative platelet counts.

The survival time in our "therapeutically" treated group is surprisingly good and none of the now surviving patients have had recurrent esophageal varix bleeding.

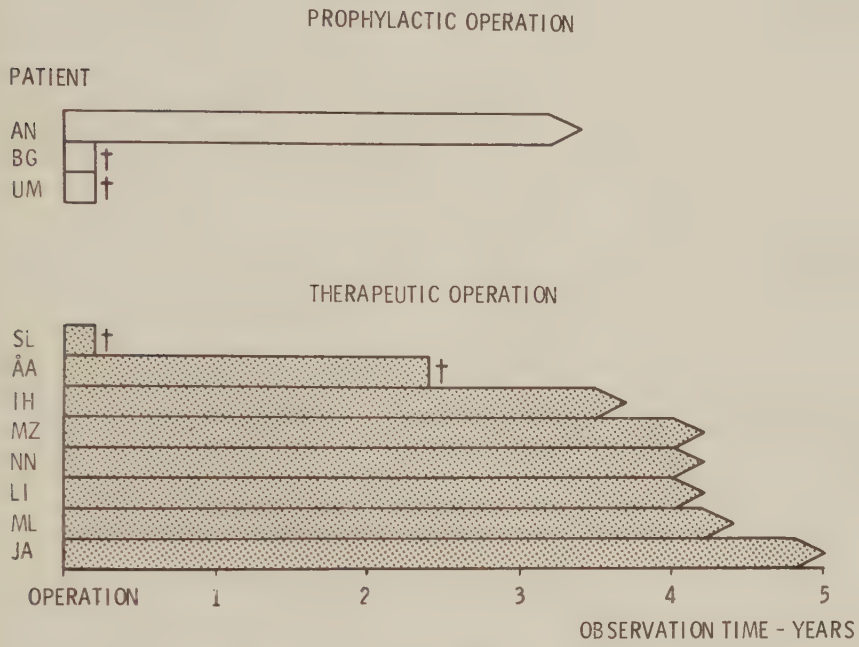

Fig. 12. Result of treatment in 11 patients subjected to subcutaneous transposition of the spleen.

Conclusions drawn from the clinical study.

- The studied operation is not without risk because of bleeding during the splenic mobilization. The operation should probably be avoided in the obese patient in whom there is difficulty for the splenic vessels to bridge the distance between their retropancreatic origin and the subcutaneous position. We regard the postoperative splenic atrophies as being a result of too much tension in these splenic vessels causing thrombosis.
- In patients with viable splenic remnants there is a collateral flow over the subcutaneously transposed spleen resulting in a reversed flow in the splenic vein but not in a decrease in portal vein pressure. Except in one patient we could not find a priority of flow in the parasplenic collaterals over that in the esophagus.

The reason why our surviving patients have done so well can possibly be attributed to a moderate collateral development that did not result in reduced portal perfusion of the liver. Considering the fact that portal hypertension remained, the frequency of rebleeding has been low. Whether this is just a matter of luck or whether it can be assigned to the elimination of hypersplenism and the development of collaterals in close vicinity to the esophagogastric varices that perhaps act as a pressure safety-valve, can only be a matter of speculation for the moment.

DISCUSSION

Survey of methods attempted for surgical treatment of portal hypertension.

A Portasystemic shunting

It is now a century since the first portacaval shunt was performed. Nicholai Eck demonstrated in 1877 the technical feasibility of this procedure in dogs and he also suggested that an anastomosis between the two veins might be used to sidetrack the venous return in obstruction of the portal vein. The first successful clinical operation of this type was reported by Vidal in 1903. He performed the operation for ascites and obtained a four-month survival. Around 1910 there were additional reports in this field. The attempts to surgical shunting between tributaries of the portal and inferior caval vein systems all suffered from the small calibre of communication resulting in early shunt failure (Villard and Travenier 1910, Gunn 1911, Muersing 1912). In 1912 there was again a successful case treated with an Eck fistula (Rosenstein 1912). However, the high mortality rates and the many shunt failures discouraged the use of shunting surgery for many years. It should be stressed that these early efforts were attempted mainly for the treatment of ascites. The portacaval anastomosis was again advocated by Mc Indoe in 1928 for treatment of complications of portal hypertension. In postmortem studies he had found that portasystemic shunts developed spontaneously in cirrhosis but appeared to be of insufficient size and located so as to predispose to gastrointestinal bleeding.
In 1945 Whipple and Blakemore and Lord reported ten patients operated with portacaval anastomoses - in five the splenic and left renal veins were united after removal of the spleen and the left kidney and in five

the portal vein was anastomosed to the inferior vena cava end-to-side. In the beginning these authors used a Vitallium tube nonsuture technique but later on direct suture. The shunting efforts were now also directed towards prevention of hemorrhages from esophageal varices since the elevation in portal venous pressure for the first time had been clearly documented and it had been increasingly clear to the discoverers that death from bleeding gastroesophageal varices was common in these patients (Thompson et al 1937). In 1947 a proximal end-to-side spleno-renal shunt was proposed by Blalock and later on specially advocated by Linton et al (1961). Longmire (1950) among others proposed a side-to-side portacaval shunt in the hope that part of the portal flow would still perfuse the liver. For extrahepatic portal obstruction Marion (1953) and later Clatworthy et al (1955) developed the mesocaval shunt by joining the proximal end of the vena cava to the side of the superior mesenteric vein.

There was a rapid growth in shunting experience and operative mortality decreased as the number of treated patients increased. Blakemore reported as early as 1952 160 shunted patients and from our own country Ekman and Sandblom reported 82 patients in 1957. The number of different types of portasystemic shunts also steadily increased - most of them being modifications of side-to-side portacaval shunts e.g. left renal vein to portal vein (Mazzoni and Martino 1973) and omphalocaval shunt (Sobel et al 1970). One of these newer shunts receiving much interest today is the interposition mesocaval shunt - H-graft (Lord et al 1971, Drapanas 1972, Smith et al 1974, Inberg et al 1974, Thompson et al 1974, Stipa et al 1976).

However, after an era of enthusiasm during the fifties and early sixties, criticism against shunting surgery was again heard. Satterfield et al concluded in 1965, that portal systemic anastomosis largely protects cirrhotic patients from recurring hemorrhage from esophageal varices but does not improve survival rate because of operative mortality and death from hepatic failure. Prevention of hemorrhage was not synonymous with increased survival. At present thousands of portacaval shunts have been conducted and the collected experience has been subjected to many reviews (e.g. Grace et al 1966, Rueff and Maillard 1974, Conn 1974 and Malt 1976). It is evident that prospective, randomized, controlled studies are mandatory to make further progress in this field. A few such studies have been done and have shed some light on the enormous and often confusing literature about treatment after variceal bleeding. There is general agreement that the portacaval shunt very effectively reduces the risk for rebleeding and is a valuable form of treatment for some patients who have bled from esophageal varices - which ones, remain to be defined. However, portacaval shunts should not be done for patients, whose varices have not bled. Under these circumstances a prophylactic shunt exchanges encephalopathy for hemorrhage without prolonging life (Editorial, Lancet, 1972). The superiority of shunting surgery over other forms of treatment after bleeding from esophageal varices is today not as self-evident as it was thought to be in the fifties and sixties. Summing up the results of four

randomized studies of the therapeutic portacaval shunt (Resnick et al 1974, Jackson et al 1973, Mikkelsen et al 1974, Rueff et al 1974) Conn (1974) states; "that it is clear that we must learn either to select better who should be shunted or shunt better those we select".

If a portasystemic shunt should be performed there is no general agreement on which type of shunt should be preferred. Voorhees et al (1970), Bismuth et al (1974), Malt et al (1976) have found no significant differences between proximal spleno-renal shunt, end-to-side or side-to-side portacaval shunts concerning postoperative mortality, long term survival, recurrence of gastrointestinal bleeding or occurrence of encephalopathy. Others (e.g. Turcotte et al 1969) have preferred the side-to-side to the end-to-side shunt in the poor risk patient because of significant lower operative mortality. Still others (e.g. Orloff et al 1974) have not been able to find this difference. Many have advocated the spleno-renal shunt especially because of lower risk of post-operative encephalopathy (Linton et al 1961, Hallenbeck et al 1963, Gill et al 1975, Pliam et al 1975). It can possibly be concluded that there is some general agreement that the side-to-side shunt relieves ascites better than the other shunts and that the proximal spleno-renal shunt possibly gives rise to a lesser incidence of postoperative encephalopathy but also a higher rate of shunt failure and recurrent bleeding. The traditional mesocaval shunt is in adults followed by a high frequency of leg oedema.

It has been evident from controlled series of both "prophylactic" and "therapeutic" portacaval shunts that the death rate from liver failure is increased after shunt. The reduced liver perfusion has been accused for this fact. Attempts have been made to overcome this deleterious effect by arterialization of the central portal vein stump. The experiences of this procedure have been reviewed by Maillard et al 1974. It has not received widespread enthusiasm but can perhaps be used for reduction of postoperative encephalopathy. Another way to reduce the drawbacks of the portacaval shunt would be a better selection of patients. For some time there was great hope that preoperative pressure and flow measurements in the portal vein system would be the solution of the problem of separating those who would benefit by the shunt from those who would not (e.g. Warren et al 1967, McDermott 1972, Smith 1973). Later this enthusiasm has been stilled and now most authors are of the opinion that such evaluations do not solve the problem (Taylor et al 1968, Reynolds 1974, Burchell et al 1974, Charters et al 1975). Lately it has been claimed that if a preoperative evaluation was possible of the capability of the patient to increase the hepatic arterial flow in response to the relief in sinusoidal hypertension produced by a portacaval shunt, this would be of major prognostic value (Burchell et al 1976).

Whether the selective decompressive procedures mentioned in the introduction will have the positive effects of conventional shunts but lack the negative ones, remains to be tested in prospective studies. In a controlled clinical study of patients subjected either to mesocaval

or to distal spleno-renal shunts, the frequency of postoperative encephalopathy was much higher after the former operation (Galambos et al 1976).

B Removal of varices and interruption of esophageal varix flow.

In 1950 Crile described a method for transthoracic, transesophageal suture ligation of esophageal varices. In 1953 he reported seven cases with esophageal bleeding secondary to extrahepatic portal block treated by this method with satisfactory results. The same year Linton and Warren advocated the same procedure for the temporary control of variceal bleeding before an elective spleno-renal shunt. In 1954 Nissen proposed suture ligatures through the esophageal wall without opening the lumen. In 1956 Welch described the transabdominal approach in patients with cirrhosis and active bleeding. The operation was otherwise the same as the one used by Crile and Linton. Ottinger and Moncure (1974) made an asessment of the method described by Crile and Linton. They reported 72 patients undergoing transthoracic ligation of varices for active hemorrhage as the immediate procedure for control of bleeding. The mortality rate was 50 percent. Twelve percent developed fistulae from the esophageal suture line. Patients belonging to Child group A seemed to benefit from the procedure but not those belonging to groups B or C.

The varix interruption can also be achieved by special ligature - resection devices as described by Vossschulte 1957, Boerema 1970 and Takasaki et al 1976. Berger and Rehbein (1973) have advocated the Vosschulte ligature as a permanent treatment of varices secondary to extrahepatic portal vein obstruction.

A form of treatment in the same field is transesophageal sclerosing injections through an esophagoscope. This method was introduced in 1939 by Crafoord and Frenckner. The method has still actuality. It has mostly been used in acute treatment or when the portacaval shunt has not been feasible. Recent reports by Hunt et al 1969, Denck 1970, Kapp and Buess 1973, Johnston and Rodgers 1973, Paquet and Engel 1974 express the opinion that sclerotherapy carries a lower morbidity and mortality than other measures for acute treatment. The sclerosing injections can also be given by slective catheterization of the coronary vein via the percutaneous transhepatic route as described by Lunderquist and Vang 1974.

The interruption of flow in the esophagogastric varices can also be obtained by other methods. After McIndoe's (1928) postmortem studies in cirrhotics it was evident that the main source of blood to the varices was through the coronary vein of the stomach. Tying that vein was tried by Roundtree et al 1929, but after a more extensive trial it was evident that the final results were not good (Walters et al 1940).

Later on different types of transections and resections were tried.

Phemister and Humphreys (1947) initiated total gastrectomy and gastro-esophageal resection for the total removal of varices. Tanner in 1950 transected the stomach through the fundus and resutured in an attempt to decrease the amount of blood going through the varices. The vasa brevia and the left gastric artery and vein were also divided. Allen reported in 1956 some additional operations of this type and added gastric decompression because of the vagotomy effect of the fundic bisection. Shafer and Kittle (1950) made resections of the lower one-third of the esophagus and the upper one-third to four-fifths of the stomach. About the same operation was reported by Womach in 1950 and Shumacker and King 1952. The assambled experience was a high frequency of rebleeding and in an effort to improve the results, Cooley and DeBakey (1954) extended the esophagus resection above the azygos vein. Merendino and Dillard were the first to interpose a segment of jejunum between the resected parts of the esophagus and the stomach(1955).Habif (1959) added to the experience in this field by reporting 28 patients. In the beginning he performed an almost total gastrectomy saving 2 cm below the cardia. The continuity was re-established by suturing a jejunal loop to the cardia. Because of subsequent rebleeding in many patients he changed the operation to a resection of the lower one-third of the esophagus also. Later on, considering the fact that the gastric varices were confined to the region below the cardia, he decided to divide the esophagus immediately below the inferior pulmonary vein and the stomach 5 cm below the cardia and to restore continuity by interposing an isolated segment of the jejunum. The patients reported by Habif were all failures after earlier treatment - most of them had had splenectomies or attempted portacaval shunts.

The intention with gastric resections in many of the above mentioned materials was not only to remove varices and interrupt variceal flow but also to diminish gastric acid secretion. Kegaris had already in 1934 speculated on the mechanism that erosion of the esophageal epithelium overlying the varices by refluxed acid-peptic juice from the stomach was quite likely. This idea was taken up by Wangensteen and Baronofsky (1949). They proposed that recurrent bleeding could be prevented by an almost total or total gastrectomy which would remove the acid-peptic factor and separate the direct communication between the portal and esophageal vein systems and also reduce the inflow of blood into the portal system. Wangensteen performed 90-100 percent gastric resection in eight patients. The mortality rate was very high. Of four survivors two rebled. In 1950 Gray and Whitsell presented an operation consisting of splenectomy, devascularization of the lower one-third of the esophagus and upper cardia along with vagotomy and gastroenterostomy. They reported three patients and no recurrent bleeding during a follow-up period of five to fourteen months. However, there are clinical observations that argue against acid-peptic erosion of the esophageal epithelium as the only or major cause of bleeding. Barnes and Redo (1957) reported a resection of the esophagogastric junction and re-established continuity with a Roux-Y isoperistaltic esophagojejunostomy, completely bypassing the stomach in four patients with bleeding varices. The three survivors all had recurrent bleeding. The results

after total or almost total gastrectomy have also disfavoured the use of acid-reducing procedures as the main form of treatment for bleeding varices.

C Reduction of blood amount entering the portal circulation.

Banti described in 1883 a disease which he believed started in the spleen and ended up with cirrhosis, ascites and death. The disease was characterized by splenomegaly and anemia. At the beginning of the century many surgeons performed splenectomy for relief of anemia and in the hope of stopping varix bleeding. Mayo reported in 1929 the results of splenectomy in 144 patients with "splenic anemia". The operative mortality was about ten percent and another ten percent died during a ten-year period postoperatively. Howells (1938) made an analysis of the treatment in 94 cases of Banti's syndrome. Fiftyone were treated by splenectomy and 43 medically. It was concluded that splenectomy does not improve the expectation of life nor does is prevent the progress of cirrhosis or of the anemia, or the occurrence of hematemesis. He found no reason to retain splenectomy as a routine. Pemberton and Kiernan described in 1945 the results in 272 splenectomies because of "splenic anemia". The operative mortality rate was 11 percent. Fourty percent of the survivors had gastrointestinal bleeding postoperatively. Of those with preoperative gastrointestinal bleeding, 54 percent had recurrent bleeding and of those without preoperative hemorrhage 15 percent bled postoperatively. Pemberton and Kiernan concluded that the effect of operation was most gratifying with a regression of the anemia and of the leucopenia followed by a general improvement of the health of the patient, as well as an improvement of the hepatic function in many of the cases. However, Rousselot (1940), Miller and Hagedorn (1951) and Shumacker and King (1952) found the rebleeding frequency too high and disfavoured the method. Rousselot discovered in 1936 that the splenomegaly in Mb Banti probably was due to congestion and not to a primary disease in the spleen. However, ever after that, splenectomies were performed in portal hypertension. The idea was both to reduce the amount of blood entering the portal venous bed by 20-40 percent and thereby reduce the pressure, and to relieve hypersplenism. Anyhow, the high rebleeding frequency condemned splenectomy especially in extrahepatic portal hypertension where it might destroy the last chance to perform a suitable shunt (Blakemore and Fitzpatrick 1951, Hallenbeck and Shocket 1955, Jekler et al 1965). However, Hallenbeck et al found in 1963 that during the first eight years after operation survival rates for patients having cirrhosis of the liver and who had bled from the gastrointestinal tract preoperatively were similar in the 106 patients treated by end-to-side portacaval or spleno-renal shunts and in 73 patients treated by splenectomy with or without omentopexy but without any type of shunt. The incidence of bleeding after operation was 66 percent after splenectomy and 25 percent after portasystemic shunts. Also Hsu (1972) found insignificant differences in survival between 48 patients treated with shunt and 61 treated with splenectomy. Mortality in the splenectomy group was mainly due to variceal bleeding

and in the shunt group to liver failure. Smith (1970) proposed splenectomy and coronary vein ligation as a first-stage procedure in cirrhotics with good portal flow. In these cases it might be a definitive treatment or postpone a portasystemic shunt. Dumont (1972) suggested that when an enlarged splenic artery can be identified by celiac angiography or at operation in patients with bleeding varices, splenic artery ligation and/or splenectomy may be reasonable alternatives to portacaval shunts. Splenic artery ligation has also later been used by Witte et al (1976) in selected patients with hepatic cirrhosis. The effect on hypersplenism was good. However, two of five patients who had the ligation for bleeding varices later required a portacaval shunt. The modern rationale of splenic artery ligation in portal hypertension is the increased splenic blood-flow found in many cirrhotics. The splenic flow has been found to exceed one liter/min in some cirrhotics (Blendis et al 1970). Lebrec et al (1976) have found that 95 percent of the splenic flow was shunted around the liver in patients who had had variceal bleeding.

The idea of splenic artery ligation as a method of treatment is not a new one. It was first performed by Blain in 1918 and the increased experience of the same author was reported in 1950. In the meantime others had reported on the method (Watson 1935, Berg and Rosenthal 1942, Everson and Cole 1948). Probably the main value of the method is that is reduces a hypersplenism in portal hypertension with lesser risk than with splenectomy. The operative mortality in connection with splenectomy in patients with portal hypertension is around ten percent in most series (e.g. Pemberton 1945, Miller 1951, Hallenbeck 1959, Hsu 1972).

In order to allow a greater inflow of blood from the portal vein through the liver, Reinhoff (1951) proposed and carried out ligation of both the hepatic and splenic arteries. Madden (1953), however, some years later rejected the method on the basis of his own poor results.

Another way for the reduction of inflow into the portal vein system was described by Peters and Womack in 1961. They had found an increased arterio-venous shunting in patients with portal hypertension. Among the viscera that appeared to be concerned particularly with this altered vascular mechanism was the spleen but also the stomach and lower esophagus. They designed an operation to control the arterio-venous fistulae in the stomach, spleen and lower esophagus. The operation included splenectomy with proximal ligation of the splenic artery, ligation of the left gastric artery, and the right and left gastro-epiploic arteries and resection of the margin of the greater curvature of the stomach. In 1969 the group reported their operative results in 55 patients with portal hypertension and bleeding varices. In 28 the operation was an emergency procedure. Twentyfive of the 55 patients died postoperatively. Besides there was a high incidence of recurrent bleeding. Hassab has used a very similar type of operation, mainly in cases of bilharzial cirrhosis. In 1970 he reported 605 such operations with an operative mortality of ten percent and a seven percent incidence

of recurrent bleeding. Sugiura and Futagawa (1973) added transthoracic esophageal transection (as described by Walker 1964), extensive paraesophageal devascularization, selective vagotomy and pyloroplasty to the method used by Hassab. They reported 84 patients mainly with postnecrotic cirrhosis. Disappearance of the varices was noted in 97 percent of the survivors and only one patient died postoperatively. Sixty patients had the operation after variceal bleeding, in 12 it was an emergency procedure and in 12 a prophylactic operation. Recurrence of variceal bleeding was noted in only two cases during the mean duration of follow-up of 30 months.

D Methods designed to facilitate the "spontaneous" development of portasystemic collaterals.

Lens described in 1892 suture of omentum majus to the abdominal wall but without affording the patient any relief. Drummond and Morison made a new contribution to this subject by their report in 1896. They had on postmortem examinations on a series of cases of hepatic cirrhosis demonstrated the presence of vascular adhesions connecting the viscera to the abdominal walls. Many of these cases did not have ascites and they proposed that an enlargement of the communications between the portal and systemic circulation in these cases prevented the development of ascites. They reported two cases. The parietal peritoneum was scrubbed with a sponge. The peritoneal covering of the liver and the spleen and the portions of parietal peritoneum opposite to these organs were specially scrubbed. The omentum was sutured across the anterior abdominal wall. For the purpose of retaining contact of the parietal with the visceral peritoneum, long, broad adhesive straps. were firmly applied circularly from the epigastrium down to the pubis. A glass tube was left in the pouch of Douglas and by frequently pumping the tube the abdomen was kept empty. After three weeks the abdomen was dry in one of the two patients and she was later on cured of her ascites. In the other patient it was unrelieved. The experience was broadened by Talma who in 1898 reported on a nine-year-old boy with cirrhosis of the liver, ascites and splenomegaly. In this patient the omentum and the gallbladder were sutured to a right-sided subcostal incision and part of the enlarged spleen was translocated through the abdominal muscle wall and suture fixated in a subcutaneous pocket. Large subcutaneous collaterals were postoperatively found originating from both scars. The boy was two years later in a very good condition and had no ascites. Morison reported in 1903 a new successful case and Talma proposed in 1904 that the omentopexy was not only of value for the treatment of ascites but might also be a valuable procedure for prevention of bleeding from esophageal varices. In 1905 Narath described his technique for omentopexy suggesting that the omentum should be brought through a little hole in the upper midline incison to a subcutaneous pocket instead of just being sutured to the inner abdominal wall. With the increased experience of the pexy operations it was found that those who seemed to benefit most from the operation were alcoholics with cirrhosis (Morison 1912). Many began to doubt the effect of the

operation and suggested that the apparent benefit was probably due to improvement in liver function related to abstinence and dietary treatment (Vander Veer 1912). In 1933 Holman, after some successful experiments in dogs, reported implantation of the spleen in the abdominal wall in one patient. He was disappointed with the results of the Talma-Morison operation. His first clinical attempt was a failure, but later on he reported two successful cases (Gray and Whitesell 1950 - discussion). The efforts with the pexy operations were continued but the main interest was directed towards thoracic transposition of the spleen after a report of three cases by the Finnish surgeons Nylander and Turunen in 1955. The method was extensively tried experimentally and clinically. Turunen and Laitinen (1959) demonstrated collateral circulation between the transposed spleen and the superior vena cava with splenoportography. Clinical reports also came from America (Hoffman and Freedlander 1960) and from France (Bourgeon 1961). Bourgeon et al (1961) described a modification of the original method - splenopexy. Part of the left diaphragm was resected to produce vascular adhesions between the superior pole of the spleen and the thoracic structures. He also introduced another modification - resection - transposition (Bourgeon et al 1962). Resection of the spleen was performed on the basis of segmental organisation of the splenic vessels. Auvert et al (1966) described spleno-pneumopexy. The decapsulated superior pole of the spleen was implanted into the left lung. The intention was the development of spleno-pulmonary anastomoses. That the latter method can result in large shunts was demonstrated by Sztaba and Sokol (1968) who in a four-year-old boy postoperatively found cyanosis and severe dyspnoea due to a wide spleno-pulmonary shunt. Since the development of spleno-thoracic collaterals takes time, the transposition has sometimes been combined with transesophageal ligation of varices (Turunen et al 1963, Auvert et al 1969). Experimental and clinical efforts concerning thoracic transposition of the spleen were reviewed by Hästbacka in 1971. He also reported the largest series - 37 patients - with the longest follow-up time (1-17 years) managed by that method. Of the 37 patients 11 had portal vein thrombosis and 26 cirrhosis. The operation was "prophylactic" in four patients. In 11 patients the spleen was resected in connection with the operation. The splenic artery was ligated at a secondary operation in 11 patients to relieve hypersplenism and reduce inflow of blood in the portal vein system. Of the patients with good or moderately impaired liver function (Sedgwick and Poulantzas 1967) one of 26 died postoperatively. Three of 11 patients with severely impaired liver function died postoperatively. Of the survivors 38 percent had recurrent bleeding during the first six months after operation. Late rebleeding occurred in 33 percent of the cases. Two-thirds of the patients had no esophageal rebleeding after the first six postoperative months. Late recurrences ended fatally in only two cases. The esophageal varices disappeared or diminished in two-fifths of the cases. This suggested - according to Hästbacka - that the developed collaterals, once formed, decompressed the esophageal varices in their vicinity even when there was no significant decrease in portal pressure. A drawback was the risk of early rebleeding. There was no detrimental effect on the liver function and the operation did not

cause hypersplenism. Hästbacka concluded that splenic transposition to the thorax was a surgical procedure that spares the patient, the use of which should be taken into consideration in many cases of portal hypertension.

Kamimura and Akita proposed in 1968 - after a ten-year-period of experimental trial - hepatopneumopexy as a clinical treatment of ascites complicating cirrhosis. The purpose was to improve the hepatic blood outflow via newly formed collateral communications. This idea was first suggested by Madden (1954) who tried to produce hepatophrenic vascular communications by the fixation of the liver to the diaphragm. Kamimura's and Akita's operation consisted of a thoracical approach, a circular resection of the diaphragm on the right side 12-13 cm in diameter, removal of the serous surface of the liver, abrasion of the diaphragmatic surface of the right pulmonary lower lobe and suture fixation of the lung, liver and diaphragm. They reported on 20 patients and found an excellent effect on ascites in 60 percent, a fair effect in 20 percent and a poor effect in 20 percent of the patients. The decrease in splenic pulp pressure caused by the operation averaged 79 mm H_2O. There were no immediate postoperative deaths. In 1968 Akita et al described splenopneumopexy in nine patients with portal hypertension secondary to liver cirrhosis and/or Budd-Chiari syndrome. The operation consisted of ligation and severing of the splenic artery, ligation of the coronary vein and fixation of the abraised surfaces of the spleen and left lower lung through a circular resected hole in the diaphragm. The procedure was found to be very effective for the treatment of the Budd-Chiari syndrome. In 1973 Akita and Sakoda described the operation in five children with portal hypertension - in one due to hepatic fibrosis and in four due to portal vein thrombosis. Four of the children had had preoperative variceal bleeding. In all cases remarkable porta-pulmonary shunts were postoperatively demonstrated by splenoportography. Splenic pulp pressure decreased adequately to prevent esophageal rebleeding. It was concluded that porta-pulmonary shunt was a safe, useful and effective surgical procedure for children with portal hypertension.

Other pexy operations than those using the spleen as the bridging organ still have their modern advocates. Berman et al (1963) described omentocavopexy on seven patients who had bled from esophageal varices. The omentum was brought through the right leaf of the diaphragm and sutured to the pericardium and diaphragm. However, four out of seven patients bled after the operation. Bader et al (1964) proposed thoracic ileopexy for portal hypertension. The procedure was effective in dogs and consisted of bringing a 20 cm segment of distal ileum on its vessel pedicle through the diaphragm. The segment was opened along its antimesenteric border, stripped of its mucose and submucosa and fixed to the denuded periostal and intercostal muscle surfaces. No reports have been traced of clinical attempts of this operation. In 1971 El-Zawahry et al reported satisfactory results on ascites of transposing the greater omentum under the subcutaneous tissue of the right side of the thorax. The operation was performed on ten patients with portal hyper-

tension due to bilharzial hepatosplenic fibrosis. The operation was shown both to form an adjuvant exit for excess hepatic and splanchnic lymph through communications between greater omentum lymphatics and those drained by the right lymphatic duct and to produce portasystemic venous collaterals. Couinaud (1973) reported additional experience of the method. He emphazised that it took some time for collateral circulation to develop. The fatal hemorrhages that could take place during this time could be avoided by the association of ligation of the esophagus over a Murphy button according to Boerema's technique.

Concluding remarks

The preceding part of this discussion - far from being a complete review - shows the extensive efforts made in this field. The proposed methods are innumerable and the conclusions drawn from them are very diverse - of course partly a reflection of the different types of disease being treated and the different objectives of the treatment. From a tentative beginning with pexy operations and splenectomies, the interest was four about two decades mainly centered on total or almost total shunting of the portal blood flow. Today there is a more doubtful attitude towards these shunts and selective shunting seems to be more attractive. Also other methods that do not cause further damage to the liver have found a re-awakened interest. It is evident that solid conclusions can only be based on prospective, controlled, randomized studies but the difficulties in connection herewith are also clear. Irrespective of the problems always met with when attempting such studies in the clinic, the number of patients needing treatment is, in this field, limited in each institution. Besides, many of the proposed methods are technically difficult yielding a high start-up cost concerning complications and mortality. The initial experience of Warren et al (1969) in 15 patients subjected to distal spleno-renal shunt included eight operative deaths. Now their operative results are quite different. In the latest reported series consisting of 22 selectively shunted patients there was no operative death and only one death during the first postoperative month (Galambos et al 1976). The distal spleno-renal shunt is accordingly not easy to perform and probably the left gastric venous-caval shunt is still more demanding technically. Even if one disregards the technical problems, there still remain patients who cannot be treated with these shunts. Warren (1974) states that the presence of clinical ascites is a contraindication to the use of the distal spleno-renal shunt as this can result in a failure to control ascites and unacceptably high mortality. Neither can the left gastric venous-caval shunt be applied in every portal hypertensive patient needing treatment. In 1975 Inokuchi et al reported that among 293 patients with portal hypertension managed by surgery during an eight-year period, the left gastric venous-caval shunt was intended in 131 and actually performed in 117.

Consequently there is still a need for alternative methods even if the selective shunts ultimately will prove their superiority in prospective randomized studies. One alternative is, of course, the conventional portasystemic shunt which, in those patients who escape its complications, represents very good therapy. Until there are safe methods to predict which patient will benefit from the shunt and which will not, the attitude towards this alternative will be hesitant.

The results achieved by Hassab with splenectomy and gastroesophageal devascularization are surprisingly good. They were obtained in patients mainly suffering from bilharzial hepatosplenic fibrosis which might be considered to be the reason for the success. However, there is a remarkable study by Sugiura and Futagawa (1973) who reported a disappearence of varices in 98 percent and a recurrence of bleeding in only two of 87 treated patients during a mean duration of follow-up of 30 months. The disease causing portal hypertension was liver cirrhosis in most of these patients and the treatment was similar to the method used by Hassab. It seems that these methods might be reasonable alternatives but again prospective controlled studies would be desirable. While we await the results of such studies, we intend to deal with the problem of complications in portal hypertension by means of trials with selective symptomatic treatment.

In those patients whose main problem is ascites and who are unrelieved by medical treatment we will continue our efforts with the peritoneovenous shunt proposed by Le Veen et al 1974. We have very limited but encouraging experience with this method.

Our assessment program for patients who have had suspected bleeding from esophageal varices includes percutaneous transhepatic portography. In connection with this investigation a selective catheterization and obliteration of the coronary vein is performed if the flow in this vein is hepatofugal. If possible, flow in the short gastric veins directed towards the esophagus is arrested in the same way. The technique is the one described by Lunderquist and Vang 1974. For the moment we use isobutyl-2-cyanobucrylate for the obliteration. The catheterization can be repeated later and possible newly developed collaterals towards the esophagus can be obliterated. Our experience so far (Lunderquist et al 1977) is that this method represents an effective alternative for acute treatment when vasopressin infusion or balloon tamponade have failed. Some patients can be given permanent relief by this method probably because of a collateralization developing in the direction of for example the left renal vein after the esophageal collateral flow has been diminished. Besides, improvement of liver function can take place with resulting decrease in portal pressure making further varix treatment unnecessary. In a few patients the method has been combined with a transesophageal sclerosing injection via an esophagoscope. This might represent an improvement of the treatment, and will be the object of future evaluation.

However, some patients cannot be permanently relieved by the described method but will have recurrent bleeding. In accordance with our search for selective symptomatic treatment our examinations of the portal hypertensive patient also include the search for hiatal hernia and gastroesophageal reflux. One patient was successfully treated two years ago with a highly selective vagotomy in combination with a transabdominal fundoplication. Besides esophageal varices, he was preoperatively found to have a gastric acid hypersecretion and a distal esophagitis. Ten years earlier he had been treated with splenectomy and two years before the described operation with a portasystemic shunt which later occluded. After that he had recurrent bleeding during two years but has following the fundoplication procedure not suffered further hemorrhages.

When hypersplenism is prominent we attack this symptom. A portasystemic shunt often leaves hypersplenism unaffected. Felix et al (1974) reported in a randomized study relief from hypersplenism in 60 percent of patients subjected to a portacaval shunt. However, in another such study reported by Mutchnick et al (1975) there was no difference postoperatively between the shunt-group and the control-group as regards hypersplenism. Furthermore hypersplenism could develop after the shunt had been applied. Even after application of a modified distal spleno-renal shunt hypersplenism was found to be unaffected (Vang et al 1977). Although splenectomy in portal hypertensive patients has been a long-condemned method , Hallenbeck (1963) and Hsu (1972) found a similar survival rate after splenectomy as after portasystemic shunt. Others (Smith 1970, Dumont et al 1972) have advocated splenectomy as a primary treatment in selected cases of portal hypertension. Dumont mentioned as an alternative splenic artery ligation. We have attempted to achieve the same effect by injection of gelfoam in the splenic artery using angiographic technique. The aim has been partial infarctions of the spleen for the relief of a pre-existing hypersplenism. This study is in progress and the method might probably remain as an alternative in selected cases. An advantage with this method in comparison with surgical splenectomy might be that the splenic vein is left untouched for later possible use in shunting surgery.

Even with selective symptomatic treatment there will remain patients who will need some form of shunt operation. In a few patients the shunting is evidently solved spontaneously in a sufficient way (Wexler and MacLean 1975) but in the vast majority this is not the case. We had hoped that after subcutaneous transposition of the spleen the collaterals could develop to a sufficient size to prevent rebleeding but that they would be small enough to avoid complications of encephalopathy and further liver damage. Although the follow-up of the patients is satisfactory concerning recurrent hemorrhages and survival, the collateral development has not been of the magnitude we have found in animal experiments. The operation is, however, good therapy for hypersplenism and the final proposal might be that subcutaneous transposition of the resected spleen should be considered in the non-obese patient with portal hypertension and with hypersplenism as the main problem.

ACKNOWLEDGEMENTS

I wish to express my warm gratitude to:

My teacher professor Stig Bengmark who initiated this study and whose enthusiasm, criticism and support enabled me to continue and finish it.
Assistant professor Tord Olin who has placed the resources of his laboratory at my disposal and has given me invaluable help and guiding advice.
Members of the staffs of the Departments of Surgery, Diagnostic Radiology, Pathology and Internal Medicine for their support and help.
Mrs Monica Radnell, Miss Ingrid Hallberg, Mrs Britt Nihlén, Mrs Ingrid Strömblad, Miss Maj-Britt Johansson, Miss Eva Kroon and Mr Sven Bergman for careful technical and clerical assistance.
Mrs Karin Nilsson, Mr Jan Strömblad and Mr Ove Bengtsson for skilful help with illustrations and printing.
Miss Monica Lundin for typing the manuscript.
Mrs Gillian Sjödahl for revising the English text.
and
Especially to my wife for all her patience and support.

This investigation was supported by grants from the Swedish Cancer Society (Project no. 98-B74-10XC and 98-B75-11X) and the Faculty of Medicine, University of Lund.

References

Akita, H., Katsuki, T., Sakoda, K., Inoshima, Y. and Takesue, M.: Treatment of Budd-Chiari syndrome with splenopneumopexy. Acta Med. Univ. Kagoshima 10:49, 1968.

Akita, H. and Sakoda, K.: Splenopneumopexy as a surgical treatment for portal hypertension in children. Acta Med. Univ. Kagoshima 15:1, 1973.

Allen, J.G. and Head, L.R.: The diagnosis of portal hypertension with notes on treatment. Surg. Clin. North Am. 36:119, 1956.

Auvert, J., le Brigand, H., Seringe, P., Michel, J.-R. et Farge, C.: Possibilités thérapeutiques de la splenopexie intra-pulmonaire dans certains cas d'hypertension portale. Ann. Chir. Thorac. Cardiovasc. 5:83, 1966.

Auvert, J., le Brigand, H., Merlier, M. et Farge, C.: Pexie spléno-pulmonaire par implantation intra-pulmonaire du pole supérieur de la rate dans le traitement de l'hypertension portale par trombo-phlébite spléno-portale. Intrathorakale Milzverlagerung bei portaler Hypertension. Schattauer Verlag, Stuttgart-New York, 1969.

Bader, K.F., Roseman, D.L., Economou, S.G. and Beattie, E.J.: Thoracic ileopexy for portal hypertension. Arch. Surg. 89:228, 1964.

Baker, L.A., Smith, C. and Lieberman, G.: The natural history of esophageal varices. A study of 115 cirrhotic patients in whom varices were diagnosed prior to bleeding. Am. J. Med. 26:228, 1959.

Banti, G.: Dell' anemia splenica. Arch. d. scuola d'anat. patol. Florence 2:53, 1883.

Barnes, W.A. and Redo, S.F.: Evaluation of esophago-jejunostomy in the treatment of lesions at the esophago-gastric junction. Ann. Surg. 146:224, 1957.

Baronofsky, I.D.: Portal hypertension with special reference to acid-peptic factor in the causation of hemorrhage and extensive gastric resection in its treatment. Surgery 25:135, 1949.

Berg, A.A. and Rosenthal, N.: Ligation of the splenic artery for thrombocytopenic purpura and congestive splenomegaly. J. Mt. Sinai Hosp. 8:382, 1942.

Berger, D. and Rehbein, F.: Hypertension portale infantile: dissection - ligature de Vossschulte. Ann. Chir. Infantile 14:405, 1973.

Berman, J.K. and Judy, H.E.: Effects of portal venous and hepatic arterial occlusion on the normal and cirrhotic liver. Am. J. Dig. Dis. 22:98, 1955.

Berman, E.J., Waite, P., Gerig, E.L. and Bakemier, R.E.: Omentocavopexy. Arch. Surg. 86:1008, 1963.

Bismuth, H., Franco, D. and Hepp, J.: Portal-systemic shunt in hepatic cirrhosis: Does the type of shunt desisively influence the clinical result? Ann. Surg. 179:209, 1974.

Blain, A.W.: Ligation of the splenic artery for Banti's disease. Report of case. Experimental studies. Surg. Gynecol. Obstet. 26:660, 1918.

Blain, A.W. and Blain, A., III: Ligation of the splenic artery, operation of choice in selected cases of portal hypertension and Banti's syndrome. Ann. Surg. 131:92, 1950.

Blakemore, A.H. and Lord, J.W., Jr.: The technic of using vitallium tubes in establishing portacaval shunts for portal hypertension. Ann. Surg. 122:476, 1945.

Blakemore, A.H. and Fitzpatrick, H.F.: The surgical management of the post-splenectomy bleeder with extrahepatic portal hypertension. Ann. Surg. 134:420, 1951.

Blakemore, A.H.: Portacaval shunting for portal hypertension. Surg. Gynecol. Obstet. 94:443, 1952.

Blalock, A.: The use of shunt or by-pass operations in the treatment of certain circulatory disorders, including portal hypertension and pulmonic stenosis. Ann. Surg. 125:129, 1947.

Blendis, L.M., Bank, D.C., Ramboer, C. and Williams, R.: Spleen and splanchnic haemodynamics in blood dyscrasia and other splenomegalies. Clin.Sci. 38:73, 1970.

Boerema, I., Klopper, P.J. and Holscher, A.A.: Transabdominal ligation - resection of the esophagus in cases of bleeding esophageal varices. Surgery 67:409, 1970.

Bourgeon, R.: La transposition thoracique de la rate. Nouveau traitement de l'hypertension portale. Presse Med. 69:733, 1961.

Bourgeon, R., Catalano, H., Guntz, M. et Domenech, A.: Les problèmes chirurgicaux posés par les tromboses et interruptions spléno-portales de l'adulte. J. Chir. 82:643, 1961.

Bourgeon, R., Catalano, H., Guntz, M. et Castellani, H.: La transposition thoracique de la rate dans certain hypertensions portales de l'enfant. Pediatrie 17:316, 1962.

Bourgeon, R. et Mouiel, J.: La chirurgie conservatrice de la rate: splénorraphie, splénectomie partielle. Presse Med. 74: 303, 1966.

Brick, J.B. and Palmer, C.D.: One thousand cases of portal cirrhosis of the liver. Arch. Intern. Med. 113:501, 1964.

Britton, R.C., Voorhees, A.B. and Price, J.B.: Selective portal decompression. Surgery 67:104, 1970.

Burchell, A.R., Moreno, A.H., Panke, W.F. and Nealon, T.F.: Hemodynamic variables and prognosis following portacaval shunts. Surg. Gynecol. Obstet. 138:359, 1974.

Burchell, A.R., Moreno, A.H., Pankre, W.F. and Nealon, T.F.: Hepatic artery flow improvement after portacaval shunt: A single hemodynamic clinical correlate. Ann. Surg. 184:289, 1976.

Charters, A.C., Brown, B.N., Sviolka, S.C., Knox, D.G. and Orloff, M. J.: The influence of portal perfusion on the response to portacaval shunt. Am. J. Surg. 130:226, 1975.

Clatworthy, H.W., Wall, T. and Watman, R.N.: A new type of portal-to-systemic venous shunt for portal hypertension. Arch. Surg. 71:588, 1955.

Cohn, R. and Blaisdell, F.W.: The natural history of the patient with cirrhosis of the liver with esophageal varices following the first massive hemorrhage. Surg. Gynecol. Obstet. 106:699, 1958.

Couinaud, C.: L'omentopexie dans les hypertensions portales. Ann. Chir. 27:855, 1973.

Collison, H.A., Rodkey, F.L. and O'Neal, J.A.: Determination of carbon monoxide in blood by gas chromatography. Clin. Chem. 14:162, 1968.

Conn, H.O. and Lindenmuth, W.W.: Prophylactic portacaval anastomosis in cirrhotic patients with esophageal varices. Interim results, with suggestions for subsequent investigations. N. Engl. J. Med. 279:725, 1968.

Conn, H.O., Lindenmuth, W.W., May, C.J. and Ramsby, G.R.: Prophylactic portacaval anastomosis. A tale of two studies. Medicine 51:27, 1972.

Conn, H.O.: Therapeutic portacaval anastomosis: To shunt or not to shunt. Gastroenterology 67:1065, 1974.

Cooley, D.A. and DeBakey, M.E.: Subtotal esophagectomy for bleeding esophageal varices. Arch. Surg. 68:854, 1954.

Crafoord, C. and Frenckner, P.: New surgical treatment of varicous veins of the oesophagus. Acta Otolaryngol. 27:422, 1939.

Crile, G. Jr.: Transesophageal ligation of bleeding esophageal varices. Arch. Surg. 61:654, 1950.

Crile, G. Jr.: Treatment of esophageal varices by transesophageal obliteration. Surg. Gynecol. Obstet. 96:573, 1953.

Denck, H.: Die Versorgung der akuten Blutung bei Leberzirrhose. Wien. Zeitschr. Inn. Med. 51:118, 1970.

Drapanas, T.: Interposition mesocaval shunt for treatment of portal hypertension. Ann. Surg. 176:435, 1972.

Drummond, D. and Morison, R.: A case of ascites due to cirrhosis of the liver cured by operation. Br. Med. J. 2:728, 1896.

Dumont, A.E., Berman, I.R., Stahl, W.M. and Stuart, M.R.: Significance of an enlarged splenic artery in patients with bleeding varices. Ann. Surg. 175:466, 1972.

Eck, N.V.: On the question of ligature of the portal vein. Voyenno Med J. 130:1, 1977. (Transl. Surg. Gynecol. Obstet. 96:375, 1953).

Editorial: The Prophylactic Portacaval Shunt. Lancet 1:999, 1977.

Ekman, C.A.: Portal hypertension. Diagnosis and surgical treatment. Acta Chir. Scand. Suppl. 222, 1957.

El-Zawahry, M.D., Ibrahim, M.S., Amer, Z., El-Maddawy, M. and El-Razek, A.A.: Omental transposition for the relief of ascites in portal hypertension of bilharzial origin. Bull. Soc. Int. Chir. 6:499, 1971.

Everson, T.C. and Cole, W.H.: Ligation of splenic artery in patients with portal hypertension. Arch. Surg. 56: 153, 1948.

Felix, R., Myerson, R.M., Sigel, B., Perrin, E.B. and Jackson, F.C.: The effect of portacaval shunt on hypersplenism. Surg. Gynecol. Obstet. 139:899, 1974.

Galambos, J.T., Warren, W.D., Rudman, D., Smith, R.B. and Salam, A.A.: Selective and total shunts in the treatment of bleeding varices. A randomized controlled trial. N. Engl. J. Med. 295:1089, 1971.

Garceau, A.J., Chalmers, T.C. and the Boston Inter-Hospital Liver Group.:The natural history of cirrhosis. Survival with esophageal varices. N.Engl. J. Med. 268:469, 1963.

Gill, S.S., Eggleston, F.C. and Singh, C.M.: End-to-side splenorenal shunt for treatment of portal hypertension. Arch. Surg. 110:254, 1975.

Grace, N.D., Muench, H. and Chalmers, T.C.: The present status of shunts for portal hypertension in cirrhosis. Gastroenterology 50: 684, 1966.

Gray, H.K. and Whitesell, F.B. Jr.: Hemorrhage from esophageal varices: Surgical management. Ann. Surg. 132:798, 1950.

Gunn, K.E.L.G.: Trans Roy. Acad. Ireland. 8:12, 1911.

Habif, D.V.: Treatment of esophageal varices by partial esophago-gastrectomy and interposed jejunal segment. Surgery 46:212, 1959

Hall, C.A.: Erythrocyte dynamics in liver disease. Am. J. Med. 28:541, 1960.

Hallenbeck, G.A. and Shocket, E.: Treatment of bleeding esophageal varices after splenectomy. Arch. Surg. 71:581, 1955.

Hallenbeck, G.A., Comess, M.S., Wollaeger, E.E. and Gage, R.P.: Bleeding varices due to cirrhosis. Survival after 1/ Nonsurgical treatment, 2/ Splenectomy with or without omentopexy and 3/ Portacaval and splenorenal shunts. Arch. Surg. 78:774, 1959.

Hallenbeck, G.A., Wollaeger, E.E., Adson, M.A. and Gage, R.P.: Results after portal-systemic shunts in 120 patients with cirrhosis of the liver. Surg. Gynecol. Obstet. 116:435, 1963.

Haney, A., Peacock, E.E. and Madden, J.W.: Liver regeneration and hepatic collagen deposition in rats with dimethylnitrosamine-induced cirrhosis. Ann. Surg. 175:865, 1972.

Hassab, M.A.: Non-shunt operations for portal hypertension without cirrhosis. Surg. Gynecol. Obstet. 131:648, 1970.

Hoffman, H.L. and Freedlander, S.O.: Supradiaphragmatic transposition of the spleen for portal hypertension. Arch. Surg. 80:452, 1960.

Holman, E.: Implantation of the spleen in the abdominal wall for portal obstruction. A suggested operation for hepatic cirrhosis. West. J. Surg. 41:255, 1933.

Hourigan, K., Sherlock, S., George, P. and Mindel, S.: Elective end-to-side portacaval shunt: Results in 64 cases. Br. Med. J. 4:473, 1971.

Howells, L.: Treatment of splenic anaemia and Banti's syndrome. Lancet 1:1320, 1938.

Hsu, K.-Y.: Portal systemic shunt for portal hypertension: Importance of bromsulphalein retention for prediction of survival. Ann. Surg. 175:569, 1972.

Hunt, P.S., Johnston, G.W. and Rodgers, H.W.: The emergency management of bleeding oesophageal varices with sclerosing injections. Br. J. Surg. 56:305, 1969.

Hällén, J. and Krook, H.: Follow-up studies on an unselected ten-year material of 360 patients with liver cirrhosis in one community. Acta Med. Scand. 173:479, 1963.

Hästbacka, J.: Thoracic transposition of the spleen for portal hypertension. A clinical and experimental investigation. Ann. Chir. Gynaecol. Fenn. 60: suppl. 176, 1971.

Inberg, M.V., Harjola, P.-T. and Scheinin, T.M.: Role of mesenterico-caval shunt in the managemant of portal hypertension. Am. J. Surg. 127:529, 1974.

Inokuchi, K., Kobayashi, M., Kusaba, A., Ogawa, Y., Saku, M. and Shiizaki, T.: New selective decompression of esophageal varices - by a left gastric venous-caval shunt. Arch. Surg. 100:157, 1970.

Inokuchi, K., Kobayashi, M., Ogawa, Y., Saku, M. and Iwaki, A.: Late results of a left gastric venous-caval shunt for esophageal varices. Analysis of 100 clinical cases. Chir. Gastroent. 9:275, 1975.

Jackson, F.C., Perrin, E.B., Smith, A.G., Dagradi, A.E. and Nadal, H.M.: A clinical investigation of the portacaval shunt. Survival analysis of the prophylactic operation. Am. J. Surg. 115:22, 1968.

Jackson, F.C.: Therapeutic portacaval shunts: The VA cooperative study after 12 years. American Gastroenterological Association Postgraduate Course on Portal Hypertension and Cirrhosis, New York, May 21, 1973.

Jandl, J.H.: The anemia of liver disease: Observations on its mechanism. J. Clin. Invest. 34:390, 1955.

Jekler, J., Toberny, Z. and Jeklerová, J.: Splenectomy in patients with extrahepatic portal hypertension: Unjustified. J. Intern. Coll. Surg. 43:589, 1965.

Johnson, G.Jr., Womack, N.A., Gabriele, O.F. and Peters, R.M.: Control of the hyperdynamic circulation in patients with bleeding esophageal varices. Ann. Surg. 169:661, 1969.

Johnston, G.W. and Rodgers, H.W.:A review of 15 years' experience in the use of sclerotherapy in the control of acute haemorrhage from oesophageal varices. Br. J. Surg. 60:797, 1973.

Kamimura, T., Sakoda, K. and Akita, H.: Clinical application of hepatopneumopexy as a new surgical treatment for ascites. Acta Med. Univ. Kagoshima 10:27, 1968.

Kapp, F. and Buess, H.J.: Ösophaguswandsklerosierung als Therapie blutender Ösophagusvarizen bei inoperablen Patienten. Vergleichenden Resultate bei 14 behandelten und 14 unbehandelten Patienten. Dtsch. Med. Wochenschr. 98:2465, 1973.

Kegaris, D.L.: The venous plexus of the oesophagus. Surg. Gynecol. Obstet. 58:46, 1934.

Lebrec, D., Kotelanski, B. and Cohn, J.N.: Splanchnic hemodynamics in cirrhotic patients with esophageal varices and gastrointestinal bleeding. Gastroenterology 70:1108, 1976.

Lens, Th.: Ned. Tijdschr.v. Geneeskunde 1:645, 1892.

LeVeen, H.H., Piccone, V.A., Diaz, C.A. and Langsam, A.A.: A porto-systemic shunt to selectively decompress esophageal varices. J. Cardiovasc. Surg. 11:21, 1970.

LeVeen, H.H., Christoudias, G., Ip, M., Luft, R., Falk, G. and Grosberg, S.: Peritoneo-venous shunting for ascites. Ann.Surg. 180:580, 1974.

Liehr, H., Grün, M., Hörder, M.-H., Binder, H. und Pöschman, A.: Portale Hypertension der Ratte durch protrahierten Gefässverschluss. Z. Gesamte Exp. Med. 155:267, 1971.

Linton, R.R. and Warren, R.: The emergency treatment of massive bleeding from esophageal varices by transesophageal suture of these vessels at the time of acute hemorrhage. Surgery 33:243, 1953

Linton, R.R., Ellis, D.S. and Geary, J.E.: Critical comparative analysis of early and late results of splenorenal and direct portacaval shunts performed in 169 patients with portal cirrhosis. Ann. Surg. 154:446, 1961.

Longmire, W.P.,Jr.: The surgical treatment of portal hypertension. California Med., 73:209, 1950.

Lord, J.W., Rossi,G., Dailiana, M. and Rosati, L.M.: Portasystemic shunts in the management of massive hemorrhage from esophageal varices due to cirrhosis of the liver. Am. J. Surg. 121:241, 1971.

Lunderquist, A. and Vang, J.: Transhepatic catheterization and obliteration of the coronary vein in patients with portal hypertension and esophageal varices. N. Eng. J. Med. 291:646, 1974.

Lunderquist, A., Owman, T., Börjesson, B. and Bengmark, S.: Isobutyl-2-cyanobucrylate in obliteration of gastric coronary vein and esophageal varices. To be published. 1977.

Lundh, B., Johansson, M.-B., Mercke, C. and Cavallin-Ståhl, E.: Enhancement of heme catabolism by caloric restriction in man. Scand. J. Clin. Lab. Invest. 30:421, 1972.

Madden, J.L.: Ligation of the hepatic and splenic arteries in the treatment of cirrhosis of the liver. Surg. Gynecol. Obstet. 96: 594, 1953.

Madden, J.L., Lore, J.M.Jr., Gerold, F.P. and Ravid, J.M.: The pathogenesis of ascites and a consideration of its treatment. Surg. Gynecol. Obstet. 99:385, 1954.

Madden, J.W., Gertman, P.M. and Peacock, E.E.: Dimethylnitrosamine-induced hepatic cirrhosis: a new canine model of an ancient human disease. Surgery 68:260, 1970.

Maillard, J.-N., Benhamou, J.-P. and Rueff, B.: Arterialization of the liver with portacaval shunt in the treatment of portal hypertension due to intrahepatic block. Surgery 67:883, 1970.

Malt, R.A., Szczerban, J. and Malt, R.B.: Risks in therapeutic portacaval and splenorenal shunts. Ann. Surg. 184:279, 1976.

Malt, R.A. : Portasystemic venous shunts. N. Engl. J. Med. 295:24, 1976 and 295:80, 1976.

Marion, P.: Les obstructions portales. Sem. Hop. Paris 29:2781, 1953.

Mayo, W.J.: Surgical aspects of diseases of lymphoid organs with special reference to spleen: with statistical report of results following removal of diseased spleen in 530 cases. Collected Papers, Mayo Clinic and Mayo Foundation, Philadelphia, 1929, W.B. Saunders Company, vol. 21, p. 631.

Mazzoni, G. and Di Martino, C.: Portal drainage into the left renal vein in patients with cirrhosis. Surg. Gynecol. Obstet. 137:487, 1973.

McDermott, W.V.: Evaluation of the hemodynamics of portal hypertension in the selection of patients for shunt surgery. Ann. Surg. 176: 449, 1972.

McIndoe, A.H.: Vascular lesions of portal cirrhosis. Arch. Patol. 5: 23, 1928.

Merendino, K.A. and Dillard, D.H.: The concept of sphincter substitution by an interposed jejunal segment for anatomic and physiologic abnormalities at the esophagogastric junction. Ann. Surg. 142:486, 1955.

Michaëlsson, M., Nosslin, B. and Sjölin, S.: Plasma bilirubin determination in the newborn infant. A methodological study with special reference to the influence of hemolysis. Pediatrics 35:925, 1965.

Miller, E.M. and Hagedorn, A.B.: Results of splenectomy. A follow-up study of 140 consecutive cases. Ann. Surg. 134:815, 1951.

Mikkelsen, W.P.: Therapeutic portacaval shunt. Preliminary data on controlled trial and morbid effects of acute hyalin necrosis. Arch. Surg. 108:302, 1974.

Morison, R.: A case of ascites due to liver cirrhosis treated by operation. Ann. Surg. 38:361, 1903.

Morison, R.: The operative cure of ascites due to liver cirrhosis (Talma-Morison operation). Proc. R. Soc. Med. 5:37, 1912.

Muersing, F.: quoted in Enderlen, Hotz and Magnus-Alsleben: Die Pathologie und Therapie des Pfortaderverschlusses. Z. Ges. Exp. Med. 3:261, 1914.

Mutchnick, M.G., Lerner, E. and Conn, H.O.: Effect of portacaval anastomosis on hypersplenism in cirrhosis: A prospective controlled evaluation. Gastroenterology 68:1070, 1975.

Nachlas, M.N.: A critical evaluation of venous shunts for treatment of cirrhotic patients with esophageal varices. Ann. Surg. 148:169, 1958.

Narath, A.: Über die sukutane Verlagerung des Omentum. Zentralbl. Chir. 32:833, 1905.

Nissen, R.: Operationen am Ösophagus. Stuttgart: Georg Thieme Verlag, 1954.

Nylander, P.E.A. and Turunen, M.: Transposition of the spleen into the thoracic cavity in cases of portal hypertension. Ann. Surg. 142:954, 1955.

Olsson, R.: The natural history of esophageal varices. A retrospective study of 224 cases with liver cirrhosis. Digestion 6:65, 1972.

Orloff, M.J., Chandler, J.G., Charters, A.C., Condon, J.K., Grambort, D.E., Modafferi, T.R. and Levin, S.E.: Comparison of end to side and side to side portacaval shunts in dogs and human subjects with cirrhosis and portal hypertension. Am. J. Surg. 128:195, 1974.

Ottinger, L.W. and Moncure, A.C.: Transthoracic ligation of bleeding esophageal varices in patients with intrahepatic portal obstruction. Ann. Surg. 179:35, 1974.

Paquet, K.-J. and Engel, C.: Die Wandsklerosierung des Ösophagusvarizenblutung und der drohenden Hämorrhagie bei dekompensierter Leberfunktion. Z. Gastroenterol. 12:395, 1974.

Pemberton, J.J., and Kiernan, P.: Surgery of the spleen. Surg. Clin. North Am. 880 aug. 1945.

Peters, R.M. and Womack, N.A.: Surgery of vascular distortions in cirrhosis of the liver. Ann. Surg. 154:432, 1961.

Phemister, D.B. and Humphreys, E.M.: Gastro-esophageal resection and total gastrectomy in the treatment of bleeding varicose veins in Banti's syndrome. Ann. Surg. 126:397, 1947.

Pliam, M.B., Adson, M.A. and Foulk, W.T.: Conventional splenorenal shunts. A reconsideration. Arch. Surg. 110:588, 1975.

Rehnström, S., Simert, G., Hansson, J.A. and Vang, J.: Chronic hepatic encephalopathy. A psychometrical study. Accepted for publ. in Scand. J. Gastroent. 1977.

Reinhoff, W.F.: Ligation of the hepatic and splenic arteries in the treatment of portal hypertension with a report of six cases. Bull. Johns Hopkins Hosp. 88:368, 1951.

Reynolds, T.B.: Promises! Promises! Hemodynamics and portal-systemic shunt. N. Engl. J. Med. 290:1484, 1974.

Resnick, R.H., Chalmers, T.C., Ishihara, A.M., Garceau, A.J., Callow, A.D., Schimmel, E.M., O'Hara, E.T. and the Boston Interhospital Liver Group.:A controlled study of the prophylactic portacaval shunt. A final report. Ann. Intern. Med. 70:675, 1969.

Resnick, R.H., Iber, F.L., Ishihara, A.M. and the Boston Interhospital Liver group.:A controlled study of the therapeutic portacaval shunt. Gastroenterology 67:843, 1974.

Rosenstein, P.: Über die Behandlung der Leber cirrhose durch Anlegung einer Eck'schen Fistel. Arch. Klin. Chir. 98:1082, 1912.

Roundtree, L.G., Walters, W. and McIndoe, A.H.: End result of tying of the coronary vein for prevention of hemorrhage from esophageal varices. Proc. Staff Meet. Mayo Clin. 4:263, 1929.

Rousselot, L.M.: The role of congestion (portal hypertension) in so-called Banti's syndrome: A clinical and pathologic study of 31 cases. J.A.M.A. 107:1788, 1936.

Rousselot, L.M.: The late phase of congestive splenomegaly (Banti's syndrome) with hematemesis but without cirrhosis of the liver. Surgery 8:34, 1940.

Rueff, B. and Maillard, J.N.: Portal-systemic shunts in patients with cirrhosis. The need of controlled clinical trials. Digestion 11:414, 1974.

Rueff, B., Prandi, D., Degos, F., Sicot, J., Degos, J.-D., Sicot, C., Maillard, J.-N., Fauvert, R. and Benhamou, J.-P.: A controlled study of therapeutic portacaval shunt in alcoholic cirrhosis. Lancet 1: 655, 1976.

Satterfield, J.V., Mulligan, L.V. and Butcher, H.R.: Bleeding esophageal varices. Comparison of operative and nonoperative treatment. Arch. Surg. 90:667, 1965.

Sedgewick, C.E. and Poulantzas, J.K.: Portal hypertension, Little Brown Co., Boston 1967.

Shafer, P.W. and Kittle, C.F.: Partial esophagogastrectomy for esophageal varices. Arch. Surg. 61:235, 1950.

Shumacker, H.B. Jr. and King, H.: Splenic studies. II. Portal hypertension in children associated with gastroesophageal hemorrhage. Arch. Surg. 65:499, 1952.

Smith, G.V.: Splenectomy and coronary vein ligation for the control of bleeding esophageal varices. Am. J. Surg. 119:122, 1970.

Smith, G.W.: Significance of hemodynamic studies in the evaluation of portal hypertension. Am. Surg. 39:40, 1973.

Smith, M., Tuft, R.J., Davidson, A.R., Laws, J.W., Dawson, J.L. and Williams, R.: Mesentericocaval "jump" graft in management of portal hypertension: Experience with 24 cases. Br. Med. J. 3:705, 1974.

Snyder, N., Reboucas, G., Brodoff, M., Conn, M.B. and Conn, H.O.: Presence or absence of esophageal varices on prognosis in cirrhosis: A prospective investigation. Gastroenterology 68:1070, 1975.

Sobel, S., Kaplitt, M.J., Popowitz, L., Girardet, R.E. and Adamsons, R.J.: Omphalocaval shunt: A new procedure for portal decompression. Surgery 68: 456, 1970.

Stipa, S., Thau, A., Ziparo, V. and Schillaci, A.: Mesenterico-caval shunt for portal hypertension. Chir. Gastroent. 10:235, 1976.

Sugiura, M. and Futagawa, S.: A new technique for treating esophageal varices. J. Cardiovasc. Surg. 66:677, 1973.

Sztaba, R. and Sokół, S.: Unerwünschte splenopulmonale Gefässanastomose nach intrathorakaler Milzverlagerung. Zentralbl. Chir. 93:1260, 1968.

Takasaki, T., Kobayashi, S., Iwatsuka, M., Suzuki, S., Hayashi, N. and Harada, M.: Treatment of esophageal varices: Our method of esophageal transection using a suture device. Chir. Gastroent. 10:137, 1976.

Talma, S.: Chirurgische Öffnung neuer Seitenbahnen für das Blut der Vena porta. Berl. Klin. Wochenschr. 35:833, 1898.

Talma, S.: Chirurgische Öffnung neuer Seitenbahnen für das Blut der Vena portae. III. Berl. Klin. Wochenschr. 41:893, 1904.

Tanner, N.C.: Gastroduodenal hemorrhage as a surgical emergency (discussion). Proc. R. Soc. Med. 43:145, 1950.

Taylor, P.C., Hertzer, N.R. and Hermann, R.E.: Differential portal pressures in relation to prognosis and to survival of patients undergoing portacaval shunt. Cleve. Clin. Q. 35:183, 1968.

Tenhunen, R., Marver, H.S. and Schmid, R.: Microsomal hemeoxygenase. J. Biol. Chem. 23:6388, 1969.

Thompson, W.P., Caughey, J.L., Whipple, A.O. and Rousselot, L.M.: Splenic vein pressure in congestive splenomegaly (Banti's syndrome). J. Clin. Invest. 16:571, 1937.

Thompson, B.W. and Read, R.C.: Interposition "H" grafting for portal hypertension. Arch. Surg. 108:502, 1974.

Turcotte, J.G., Wallin, V.W. and Child, C.G.: End to side versus side to side portacaval shunts in patients with hepatic cirrhosis. Am. J. Surg. 117:108, 1969.

Turunen, M. and Laitinen, H.: Collateral circulation between a spleen transposited into the thoracic cavity and the vena cava superior. Ann. Surg. 149:443, 1959.

Turunen, M., Pasila, M. and Sulamaa, M.: Transponierung der Milz in die Brusthöhle zur Behandlung von durch Pfortaderstauung verursachten Oesophagialblutungen, II. Weltkongress für Gastroenterologie, München 1962; III: 478, 1963.

Vander Veer, E.A.: Talma operation for cirrhosis of the liver. Surg. Gynecol. Obstet. 15:278, 1912.

Vang, J., Simert, G., Hansson, J.-A., Tylén, U. and Bengmark, S.: Results of a modified distal splenorenal shunt for portal hypertension. Accepted for publ. Ann. Surg. 1977.

Vidal, N.: Traitment chirurgical des ascites. Presse Med. 2:747, 1903.

Villard, E. and Tavernier, L.: Suture ovario-mesentérique dans un cas de cirrhose du foie. Lyon Med. 114:1113, 1910.

Voorhees, A.B., Price, J.B. and Britton, R.C.: Portasystemic shunting procedures for portal hypertension. Twenty-six year experience in adults with cirrhosis of the liver. Am. J. Surg. 119:501, 1970.

Voorhees, A.B.: Portal hypertension as I see it. In Portal Hypertension. Chapter three. Ed. Child. C.G. W.B. Saunders, Philadelphia. 1974.

Vossschulte, K.: Dissektionsligatur des Ösophagus bei Varicen der Speiseröhre infolge Pfortaderhypertonie. Chirurg 28:186, 1957.

Walker, R.M.: Esophageal transection for bleeding varices. Surg. Gynecol. Obstet. 118:323, 1964.

Walters, W., Moersch, H.J. and McKinnon, D.A.: Bleeding esophageal varices: An evaluation of methods directed toward their control, especially direct injection of a sclerosing solution. Arch. Surg. 41:1101, 1940.

Warren, W.D., Zeppa, R. and Fomon, J.J.: Selective trans-splenic decompression of gastroesophageal varices by distal splenorenal shunt. Ann. Surg. 166:437, 1967.

Warren, W.D., Fomon, J.J., Viamonte, M. and Zeppa, R.: Preoperative assessment of portal hypertension. Ann. Surg. 165: 999, 1967.

Warren, W.D., Fomon, J.J. and Zeppa, R.: Further evaluation of selective decompression of varices by distal splenorenal shunt. Ann. Surg. 169:652, 1969.

Warren, W.D., Salam, A.A., Hutson, D. and Zeppa, R.: Selective distal splenorenal shunt. Arch. Surg. 108:306, 1974.

Watson, R.B.: Ligation of splenic artery for advanced splenic anemia. Br. Med. J. 1:821, 1935.

Welch, C.S.: Ligation of esophageal varices by transabdominal route. N. Engl. J. Med. 255:677, 1956.

Wexler, M.J. and Mac Lean, L.D.: Massive spontaneous portal-systemic shunting without varices. Arch. Surg. 110:995, 1975.

Whipple, A.O.: Problems of portal hypertension in relation to the hepatosplenopathies. Ann. Surg. 122:449, 1945.

Witte, C.L., Witte, M.H., Renert, W., O'Mara, R.E. and Lilien, D.L.: Splenic artery ligation in selected patients with hepatic cirrhosis and in Sprague-Dawley rats. Surg. Gynecol. Obstet. 142:1, 1976.

Womach, N.A.: Discussion paper by Gray and Whitesell. Ann. Surg. 132:808, 1950.

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